THE CLINICS
OF
JOHN B. MURPHY, M.D.

Mercy Hospital Medical Library
Chicago

VOLUME III *NUMBER 5*

THE CLINICS

OF

JOHN B. MURPHY, M.D.

AT

MERCY HOSPITAL, CHICAGO

Mercy Hospital Medical Library
Chicago

OCTOBER, 1914

PHILADELPHIA AND LONDON
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PUBLISHED BI-MONTHLY (SIX NUMBERS A YEAR), BY W. B. SAUNDERS COMPANY, WEST WASHINGTON SQUARE, PHILADELPHIA. PRICE PER YEAR: $8.00; FOREIGN, 35 SHILLINGS. BOUND IN CLOTH, $12.00 PER YEAR

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MURPHY'S CLINICAL TALKS ON SURGICAL AND GENERAL DIAGNOSIS

A DIAGNOSTIC TALK ON THE SYMPTOMS AND SIGNS OF RENAL AND URETERAL STONE, ILLUSTRATED BY A CASE

HISTORY

THE patient is an unmarried man, aged thirty-eight years. His family history is negative. His past history is negative except for rheumatism, which he had in 1907, and which laid him up in bed for six weeks.

The patient has been otherwise perfectly well until Wednesday, April 15, 1914. At about 8 A. M. on that day he was suddenly taken with a severe, sharp pain in the left loin, which lasted about thirty minutes. During this attack of pain he felt nauseated, but did not vomit. The pain was steady and continuous, and did not radiate or change its location. He felt well the rest of the day, except for slight weakness. He noticed no further symptoms until nearly midnight on Friday, April 17, 1914, when he was awakened with the same pain in exactly the same location as the previous attack. This pain lasted twenty minutes. After it stopped he became nauseated and vomited once. He felt well Saturday until 2 P. M., when another attack came on suddenly the pain being more severe than at any previous time. He became nauseated and vomited. The pain continued severe until Sunday noon, and then it became somewhat less and seemed lower down. On Sunday evening, April 19, he noticed tenesmus of the bladder for the first time. Saturday and Sunday he had frequent urinations, and thinks that on Sunday the pain radiated into the left thigh. He never passed blood at any time, and, so far as he knows, had no chills or fever.

COMMENTS

DR. MURPHY (April 20, 1914): The patient came into the hospital complaining of severe and intense pain in the left side, coming on suddenly without a moment's warning.

There is no blood in the patient's urine today, and none has been found since he entered the hospital. This attack occurred on Saturday night. Today is Monday. He had immediately, on entering the hospital, one physical sign which is very indicative of distention of the renal pelvis—intense pain on fist percussion over the left lumbar region. This symptom was never more beautifully shown than by this patient, and I could not demonstrate it in a case where it could have had greater diagnostic value, because of the vagueness of the other focal symptoms and because of the absence of blood in the urine, which is so commonly an important diagnostic point in renal and ureteral calculus. This symptom was present just at a time when there was no other physical sign of localizing value. Where there is an acute infection of the kidney, with a sudden distention of the renal pelvis, or capsule, there is always, on deep fist percussion, the same very severe pain. This infection may be either ascending, a pyelitis or pyelonephritis, or hematogenous, an embolus. I think I have never been so much impressed with the value of the test, as in this case. I have been enthusiastic about it for years, but in the other cases we have always had some confirmatory evidence pointing to the kidney as the seat of trouble, when we came to examine the urine. Here we had nothing of confirmatory value from the laboratory, and so we had to rely entirely on the history and this one physical sign, which, when so beautiful and clean-cut as in this case, aids one greatly in differential diagnosis. It is especially valuable in differentiating between the three common sites of lesions occurring acutely in the right side of the abdomen, the gall-bladder, the appendix, and the kidney. Where the test is positive on the left side of the abdomen, one can differentiate between pathology in the pancreas and in the kidney on that side. One of the great advantages of this test is that it often enables one to make the correct diagnosis of a case at the patient's bedside, without the delay incident to laboratory examinations; and, therefore, it conduces to prompter and more efficient therapy. See how this case contrasts with the case of perinephritic abscess we recently had! (See this issue of the Clinics.) That woman had a clean-cut history of an infection a month after an abortion, a chill preceding the pain, with few leukocytes in the urine, local sensitiveness in the loin, and the continued, relatively high temperature of a severe infection. This man had his pain first, than a *slight* elevation of temperature. He also had that peculiar chilly feeling common with severe pain; but he did not have a genuine severe chill. Laboratory examination failed to reveal any blood in his urine, a negative feature rather characteristic of a stone starting to pass down the ureter. This is Monday. The attack set in on Saturday night. The patient still has pain this morning, but the pain has crept downward and now is situated just at the brim of the pelvis, on the left side. The stone has, therefore, been working downward in the ureter, and will probably soon pass into the bladder, and *then* blood will appear in the urine. If the stone cease to advance, the colic will stop. The pain of ureteral stone is a colicky pain. Colicky pain, however, is produced not only by the passage of a renal calculus, but also by a fecal concretion or a hepatic calculus. Whatever the variety of the calculus, so long as it advances it causes pain, and when it stops, the pain ceases.

The causation of pain by a calculus also predicates, as a rule, its being large enough to cause some degree of obstruction to the lumen in which it lies. A stone may remain for weeks or even years in the ureter without the recurrence of a colic. The pain returns when the stone starts moving. That is a feature of great diagnostic moment which we must constantly bear in mind.

NOTE.—The patient was put on medical treatment and remained in the hospital for about two weeks without having a further attack. He then returned home, and has had no trouble since. He passed no stone and no blood during his stay in the hospital. The stone is probably suspended somewhere along the ureter. Repeated *x*-ray examinations were negative, the patient being very fat, and the stone probably a uric-acid calculus and small.—ED.

THE DIFFERENTIAL DIAGNOSIS OF THE CAUSES OF HEMATURIA. ROUTES OF METASTASIS AND CLINICAL COURSE OF MALIGNANT TUMORS OF KIDNEY AND TESTIS

HISTORY

The patient, a housewife aged sixty, entered the hospital originally February 12, 1912, with hematuria as her chief symptom.

Her family history is negative as to malignancy and tuberculosis. She never has been associated with a tuberculous person to her knowledge. Her menstrual history is normal. She had the menopause at the age of fifty, and has never menstruated since. She has six living, healthy children.

In September, 1911, five months ago, she began to have pain at times in the right lumbar and iliac regions. This pain bore no relation to the menses, which had stopped long before. It was sharp and aching, and radiated down the thighs. It never became acutely agonizing or colicky at any time. The urinations were accompanied by pain, and the amount of urine was increased, the patient going to the urinal several times every night. In September, 1911, a small amount of blood was voided with the urine for the first time. The amount was less than half a glassful. The passing of blood, together with the pain, caused her to consult a doctor.

In November, 1911, she had a very severe attack of pain in the middle of the night and vomited several times. In January, 1912, about the first of the month, she passed enough blood *per urethram* to fill a quart bottle. Since then she has been so weak that she has stayed in bed constantly.

Her right iliac region and the region just above and to the outside of it are the seat of the greatest tenderness and pain. A tumor mass is present in this location. She had a chill one week before entering the hospital, but does not know as to fever. Her temperature was normal on entering the hospital.

Examination of the chest reveals a well-marked pigeon breast. The heart is free from pathologic findings.

Cystoscopic examination on February 13, 1912, showed very little congestion about the opening of the right ureter, but a blood-clot could be seen escaping from it. There was no erosion or congestion of the left ureteral opening. It was decided that there was not sufficient evidence at the right ureteral opening to justify a diagnosis of suppuration of the kidney. The ureters were then catheterized, and 20 c.c. of urine was obtained from each one. The urine from the right side was much the more cloudy. The laboratory findings were as follows:

Urine from the left kidney: Numerous red cells are present —about 30 to a one-sixth objective field; leukocytes, 6 or 7 in a one-sixth objective field; a few granular casts are also present.

Urine from the right kidney: Erythrocytes, from 12 to 16 in a one-sixth field; leukocytes, from 4 to 6 in a one-sixth field; no casts were found.

Cultures showed the presence of *Staphylococcus albus* in the urine. On repeated examination the tubercle bacillus was not found.

A blood count showed 8,000 leukocytes; erythrocytes, 3,040,000; hemoglobin, between 60 and 70 per cent.; blood-pressure, 110 mm. of mercury.

An examination of the voided urine made on February 12, 1912, showed a small amount of albumin; no sugar; a few finely granular casts; an occasional red blood-corpuscle; a few white blood-cells; some vaginal epithelium. The specific gravity was 1015; the reaction, acid. Examination of the urine on the thirteenth of February was of the catheterized specimens reported above. An agar slant was inoculated with sediment from the left kidney and showed numerous colonies after eighteen hours. The urine from the right kidney showed only three colonies. Examination of the colonies showed a pure culture of *Staphylococcus pyogenes albus*. Examination of the urine on February 15 showed a specific gravity of 1030; an acid reaction; albumin present; no casts; no sugar; numerous red blood-corpuscles; few white blood-corpuscles; some vaginal epithelium.

The patient was operated on February 14, 1912. An incision was made below the twelfth rib on the right side, passing downward, and forward. The kidney was grasped and pulled into the field, and the pelvis of the kidney was exposed. The proximal end of the ureter was found to be dilated. Pulsating vessels were found running into the pelvis of the kidney and in front of the ureter just where it comes out of the pelvis. The vessels and ureter were ligated and cut. The kidney was found to be enlarged and nodular. It was freed from the surrounding structures and removed. One rubber drainage-tube was inserted to the bottom of the wound, and the skin closed about it in the usual way.

Examination of the excised kidney showed it to contain large tumor masses, almost caseous in appearance, near the pelvis and also in the cortex. Grossly, the tumor looked like a hypernephroma. The tumor was sent to the laboratory for section and microscopic diagnosis, and was reported by Dr. Zeit to be a hypernephroma.

The stitches were removed on February 23, but the tube was left in. There was a serosanguineous discharge present, without urinous odor. The tube was removed on March 1.

The patient entered the hospital again on March 27, 1914. She tells us that since the operation in February, 1912, up to the present time she has had dull, continuous pain all through the right side of the abdomen and chest and also in the right arm and right leg, extending in the lower limb along the inner side of the thigh clear to the foot. Both the arm and leg feel numb and weak. About February 1, 1914, she had a sudden severe attack of sharp pain, referred to the bladder and urethra, while trying to urinate. She could not void the urine, so she put a hot-water bottle over the bladder and the urine passed at once. At the same time she voided a small clot of blood. She has passed no blood since that time. She had no chills or fever at this time and has had no pain in bladder or urethra since then. However, she has had sharp, irregular pains, shooting in character, in the right side of the abdomen and in the lumbar region, radiating posteriorly beneath the scapula and into the right shoulder. About one week after the onset of the sharp pains referred to above (*i. e.*, February 8) she began to expectorate blood mornings, and continued to do so for about two weeks. About March 1, 1914, at midnight, she awoke suddenly and was very dizzy. She could not stand and felt very weak. Immediately thereafter she had emesis, but no blood was noted in it. She had emesis three or four times during the night, but not since then. At times, when turning from the right side over to the left, she has a sensation as if a mass in her abdomen moves to the left, and then back again to the right when she turns back. She has had no actual chills, but often has chilly sensations. She has had no unusual perspiration. She knows nothing as to her temperature, and has lost but little weight.

Urine examination: Specific gravity, 1020; no albumin; no sugar; an occasional granular cast; about 30 white cells to the field of a one-sixth objective; no red cells.

A white cell count showed 9400 leukocytes.

COMMENTS

DR. MURPHY, April, 1914: Let us have again the history preceding the first operation.

(Intern reads the history.)

DR. MURPHY: That is a misstatement, I believe, as to the amount of urine being increased. The last statement regarding increased frequency does not justify the conclusion that the amount was increased, as the intern who wrote the history appears to have thought, because urination often is increased in frequency, but the amount of urine not increased. The amount may be diminished and yet the frequency increased. The intern had no right to draw that conclusion from the patient's story unless he has actually measured the quantity and knows it to be increased.

The cystoscopic examination in February, 1912, showed us pretty definitely from which kidney the blood came. It was from the right side. Further, there was no redness, congestion, or edema about either ureteral orifice. The examination of the separated urines obtained by ureteral catheterization was misleading, more blood appearing from the left than from the right ureter as a result of the trauma to the left ureter by the catheter. It is this accidental hemorrhage due to slight tears of the ureteral mucosa which compels one to be so careful about interpreting the presence of blood in specimens of urine obtained by ureteral catheterization. But we actually saw the blood coming from the right ureteral orifice before we introduced the catheter. Therefore it was easy to tell that the right kidney was the one which had been bleeding. It was on this side that the tumor lay. This complaint is very similar to what? To her present complaint, except that she had her previous hemorrhage during the attack of pain. She had the first pain in September, 1911, and the hemorrhage in November. That hemorrhage was of such large quantity that it exsanguinated her, ran the erythrocytes down to 3,040,000, with a hemoglobin of from 60 to 70 per cent. That gives an index of 0.8 +, which is about right for a secondary anemia of moderate degree.

What is the most common cause of bleeding from the kidney? It is a stone. Now, although she had a history of colic at this particular time, she had a very profuse hemorrhage, which is not commonly the case with stone. With stone you may have the blood in sufficient amounts to see it with the naked eye, but you do not commonly have it in such quantities that it fills a basin. She has no colic except the colic at the time of the bleeding, just as now, which means what? That the blood is passing in clots through her ureter. I will show you later another case which belongs in this class, and which will give you a good picture of the class.

Another condition which frequently causes blood in the urine is renal tuberculosis. It is usually associated with frequent urination, which is commonly the primary symptom. You will remember the intern said she had a larger quantity of urine, a point which we did not concur in, because we said he had no right to make such a statement. Then we made the tuberculin test and a guinea-pig injection, both of which were negative. We examined the urine repeatedly for tubercle bacilli, but none were found. That is not at all uncommon with tuberculosis, especially with cortical tuberculosis of the kidney. Remember, you can have great hemorrhages with cortical tuberculosis where the tuberculous ulcer is not opening into the renal pelvis and no bacilli are present. You are less likely to have hemorrhage when the tuberculosis is in the renal pelvis. Kidney tuberculosis may begin in any one of three positions: first, the cortex; second, the pyramids; third, the calices or mucous surface of the pelvis of the kidney. With the lesion in any one of these three locations, you may have increased frequency of urination. You never find tubercle bacilli unless there is an opening between the focus and the pelvis of the kidney, and so the tubercle bacilli in the urine may be a very late manifestation of tuberculosis of the kidney, just as it is often a very late manifestation of tuberculosis of the lung. But you have the tuberculin test, which, with an active tuberculosis to the degree of ulceration, should give a high febrile reaction the next day. That test was negative. That excluded tuberculosis. Still further to exclude a tuberculosis, you have the appearance of the ureteral opening at the cystoscopic examination. There was no congestion at its mouth or at the internal urethral orifice, as is so commonly present with tuberculosis.

Malignant tumors of the kidney are a very common cause of hemorrhage. First in frequency is the hypernephroma; next, the sarcoma; next, the carcinoma. Then you can have hemorrhage from the more innocent types of tumor which occur in the kidney.

Now, to come back to the patient: We told you what we did in the first operation. She says she has pain now in the right side of the abdomen, a little in front and a little behind. But she has the most pain in the upper right quadrant of the abdomen. That also is where she is most sensitive.

Now, you might think of a kidney tumor, but the right kidney is out in the laboratory. That is the point I want to make. Therefore the tumor cannot be in her right kidney. In this connection I should like to mention another case. Have you the history of the physical findings in the abdomen in Dr. Golden's case of sarcoma of the testicle?

INTERN: There were no positive physical findings in the abdomen in Dr. Golden's case; nothing was found until the operation.

Dr. Murphy: I want to bring in that case because it is a beautiful one to contrast with this. Dr. Golden's case had the lesion on the left side. Read the history.

HISTORY (Dr. Golden's Case)

Patient, male, aged fifty-eight, married, entered the hospital March 13, 1914.

No tuberculosis or cancer in the family; wife has had no miscarriages. Patient had typhoid fever six weeks ago. He was operated twenty-three years ago for hemorrhoids. He denies all venereal disease.

About June 1, 1913, he first felt the left testicle very hard and slightly larger than the right. It could be squeezed firmly without producing any pain at all. It continued to grow larger until three months ago, when it reached the size of a hen's egg and was somewhat flattened. He consulted a doctor, who advised its removal, but made no diagnosis. Since that time it has increased more rapidly in size, so that now it is the size of a large orange, somewhat flattened and nodular, but not adherent to the skin. About February 1, 1914, the left inguinal glands became enlarged and at times are painful, especially in walking. There is no involvement of the other testicle, and there has been no bladder or urinary trouble at any time. His bowels are not constipated.

An incision was made into the enlarged testicle and a specimen sent to the laboratory for diagnosis on March 14. It was diagnosed as a large round-cell sarcoma, with perhaps a trace of alveolar arrangement.

On March 15 he was again operated and the left testicle enucleated and sent to the laboratory to confirm the diagnosis of sarcoma. The scrotum was closed without drain. An anterior incision was then made over the left renal area and the field inspected for enlarged glands. These were found and removed and sent to the laboratory. The glands showed extensive metastases. A rubber drain was inserted.

Dr. Murphy: How large were these glands?

Intern: Just about the size of a walnut; and they were situated just below Poupart's ligament.

Dr. Murphy: How many were there?

Intern: I do not remember.

Dr. Murphy: There was no tumor in the abdomen?

Intern: No tumor was felt in the abdomen.

Dr. Murphy: What was the doctor's diagnosis? That it was a sarcoma. If it was a sarcoma, why should he have an enlargement of the glands in the groin? Sarcoma is not transmitted through the lymphatics, as a rule. It is transmitted chiefly through the blood-vessels. That is the rule. The exception to the rule is the left testicle. Think of it! It shows you how important it is to know. From the left testicle the sarcoma cells may be transmitted through the lymphatics and appear in the lymph-glands below Poupart's ligament; but the most common place in which they are arrested in their spread from the left testicle is in the gland on the left renal vein. Unless you go after that gland there is no use in doing an operation for the removal of a testicle sarcoma which has been present so long as this one was present. When Dr. Golden took out the testicle and saw there was a sarcoma, he did a laparotomy at once and took out the glands which were situated on the left renal vein. You can see that an amputation of that man's testicle alone would not be of any benefit to him. A removal of the glands on the anterior surface of the renal vein was of the utmost importance in the attempt to save his life. He had no urinary findings, no evidence of an abdominal mass, thus showing the bladder was not involved by the affection; and yet, from our clinical experience, Dr. Golden felt that the renal glands were probably involved in this type of tumor, opened the abdomen, found and removed them.

Let us come back to this case before us. This woman had a hypernephroma of the right kidney. The kidney was removed. Hypernephromata do not, as a rule, attack the glands in the neighborhood, but do occasionally pass into the more distant lymphatics, and can be arrested in the glands into which these drain. More frequently, however, metastasis takes place by the blood-stream in distant organs, first in the lung, but also commonly in the other kidney. Sarcoma almost always recurs in the other kidney if the patient lives a period of three years. There are only a few authentic exceptions to this rule in the entire literature of sarcoma of the kidney; and, remember, we included hypernephroma with sarcoma of the kidney until very recently. Within the last third of a century and until the last ten years hypernephroma was called sarcoma. When I looked up the literature some ten years ago there was not a single authentic case of sarcoma of one kidney which did not recur eventually in the other. That fact suggests what? The recurrence of this hypernephroma on the right side, either in the connective tissue or in the blood-vessels. You will often find a protrusion of the tumor into the renal vein. In hypernephroma which begins in the pelvis of the kidney you frequently find extension into the renal vein. It is easy to put on a clamp and cut off the renal hypernephroma and yet leave that portion of the tumor which is in the vein. You cannot follow the tumor any farther at operation. You often have secondary manifestations in the lung, but recurrences in the other kidney are almost equally common manifestations. Whether it is by lymphatic transmission or blood transmission we do not know, but the clinical fact is that it occurs in the other kidney. It occurs also in the liver, the spleen, the bones, and in other tissues.

Now, this patient has commenced to have the same train of symptoms from the left kidney that she had originally with the right; but in addition she has what? The expulsion of blood-clots. She has blood in the urine. She has colic. She will run along probably for from one and a half to two years and, finally, depending on the size of the growth, she will die from her hypernephroma; but you want to know the pathologic course of the disease in order to know what to do. She came in today for a removal of the tumor on her left side. It will not do to go in and take out that tumor on the left side because of the bleeding from the kidney. The bleeding is not from the right side. It is from the left side; so a radical operation of any type would be positively unjustifiable so far as this condition is concerned. That is one of the reasons for having her here this morning, to impress on you the clinical course of these cases, and to show you that your knowledge of the natural clinical course aids you in keeping this patient out of additional suffering from mistaken surgery. You have to give an unfavorable prognosis, but then you did not make the hypernephroma, and that it means death to the patient is not your fault. That is a beautiful case to work out, but a few such cases are enough for a lifetime.

A recent thoroughgoing study of the pathology, pathogenesis, and routes of metastasis in hypernephroma which I can recommend highly to those surgeons who are so fortunate as to have a reading knowledge of German is that of Kostenko, "Zur Kenntniss der Hypernephrome," in the Deutsche Zeitschrift für Chirurgie for 1911, vol. cxii, pp. 284–367. The recent theories as to the origin and nature of malignant tumors of the kidney are subjected here to a thorough discussion based on a splendid material and a painstaking consideration of all the related details.

[May 8th: No subsequent operation or death yet.]

INTRATHORACIC SARCOMA STARTING FROM THE VERTEBRAL COLUMN.—DIFFERENTIAL DIAGNOSIS

EXAMINATION AND DISCUSSION BY DR. CHARLES L. MIX

HISTORY

The patient, a married man, aged forty-seven years, entered the hospital April 26, 1914, with the following history: His family history is negative to tuberculosis. His wife and five children are living and well. His wife has had no miscarriages. The patient's previous history is negative. He denies venereal infection.

The patient's present illness began as follows: About December 1, 1913, he began to have coughing spells which would last about ten minutes, as a rule. This cough was dry and unproductive. The paroxysms of coughing were especially prone to occur after retiring for the night, and they often kept the patient from sleeping, because of their severity and frequency. This condition continued until about Christmas, 1913, when he noticed, for the first time, pain in the upper right quadrant of the abdomen, dull in character. The day after Christmas the pain became worse and radiated from the upper right quadrant of the abdomen to the epigastrium, and also around to his back and up under the right scapula, then passing diagonally up the chest. This pain has always been increased by deep inspiration. In the days following the onset of the illness the pain in the hypochondrium remained dull in character, with occasional stabbing sensations, always radiating upward and toward the posterior side of the thorax These stabbing pains are increased in severity when the patient raises his right arm. The pains have never radiated downward toward either the lower abdomen or the thigh. They have never been cramp-like or colicky in character. They bear no relation to the taking of food, to the bowel movements, or to urination. On the second day after the pain came on, the patient noticed great tenderness in the right upper quadrant of the abdomen. The area of tenderness, like the pain, also extended to the epigastrium, and, on the right side of the body, as far posterior as the vertebral column. This tenderness remained fairly constant during the ensuing days and weeks. The pain would often remit somewhat, but always came on with full severity whenever the patient lay down. For this reason he has slept sitting up in a chair for the last two weeks.

His cough during the weeks following the onset of his illness remained as previously described—spasmodic and unproductive. On January 18, 1914, after a severe spell of coughing, he vomited and noticed about one dram of bright-red blood in the vomitus. He does not know for certain, however, whether this blood came from the lungs or from the stomach. The first bowel movement following this spitting of blood was also stained with bright-red blood. Since the first blood appeared he has often raised slight amounts of blood from the throat on other occasions. He has had piles for the past ten years, which have been especially troublesome during attacks of constipation, and the patient has often noticed blood on the toilet paper at such times. He thinks himself that the blood which appeared in the stools following his first bloody emesis may have come from the piles, since he was severely constipated at that time.

For the past few years the patient has had "indigestion" and "heart-burn," the attacks coming on from one-half to one hour after meals. He belches a great deal of gas. He has vomited, however, only once as above described.

On February 23, 1914, he was operated on (at another hospital) in the belief that his symptoms were due to gall-stones. No stones were found. Adhesions between the gall-bladder and intestines were reported present. The doctor told him on entering the hospital that he was jaundiced, but the patient and the patient's family say that they never noticed any jaundice. The doctor found him extremely tender over the gall-bladder on perpendicular hammer percussion. A drain was put in the wound at the operation and left in for three weeks. Six weeks after the operation the wound was healed completely, but the pain, tenderness, and cough remained the same as before the operation. The patient has never had any clay-colored stools, nor any chills, fever, or night-sweats. He has never collapsed, fainted, or suddenly felt weak or turned pale. He has never had any symptoms pointing toward urinary trouble.

Examination.—Tenderness is marked over the gall-bladder region and below the ribs on the right side. No rigidity is present in the abdomen. Some râles can be heard over the lower lobe of the right lung. There is no sharply localized point of tenderness in the abdomen. The lower right chest, both anteriorly and posteriorly, bulges slightly and the spinous processes of the vertebral column deviate slightly to the left at the same level. Fist percussion over the right kidney is negative, but over the vertebral deviation and on pressure the spinous processes produce great pain, extending along the ribs to the gall-bladder region. The patient has lost 40 pounds in weight since February 23, 1914.

The *x*-ray plates show a definite, circumscribed, rather uniformly dense mass in the lower part of the right lung, just above the diaphragm, overlapping and apparently closely connected with the vertebral column.

Examination of the blood, made on April 27, 1914, showed 11,200 leukocytes, and hemoglobin between 70 and 80 per cent.

DR. MIX (April 30, 1914): The last point mentioned in the intern's history is one of huge importance, namely, that the patient has lost 40 pounds in weight since the twenty-third day of February, 1914. Such a great loss of weight is invariably indicative of infection, hyperthyroidism, or malignancy. Since this patient is running little or no temperature and shows no signs of infection, and since he has no enlargement of his thyroid gland or other symptoms of hyperthyroidism, the suspicion of malignant disease is immediately aroused.

Before analyzing his history there are one or two statements which must be dismissed as being totally irrelevant. One of these is the history of indigestion and heart-burn which the patient says he has had for the past few years, associated with a good deal of belching of gas. We have been unable to find out that these things are in any way related to his present malady. We must also get out of the way another statement in the history which might be the source of some confusion. It will be remembered that on the occasion of his spitting up some blood in January, 1914, he had on the next succeeding bowel movement manifestations of blood in the stools. At this time the patient was very much constipated, and knowing that he had had piles for ten years' time, it was perfectly evident that the blood, which was bright red in character, being passed at a time of constipation in a patient who was then troubled with piles, clearly came from hemorrhoids.

We are now in a position to analyze his symptomatology. The patient is forty-seven years of age, with a negative venereal history, having five living children and his wife having had no

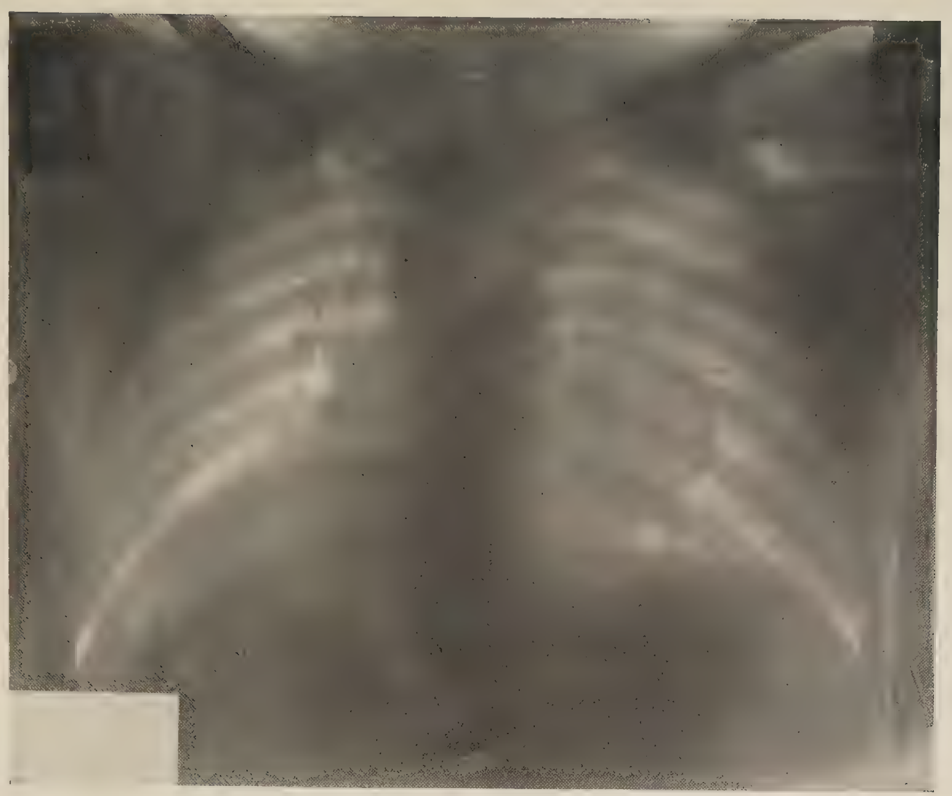

Fig. 269.—Sarcoma in the lower right chest probably originating from the spinal column and involving the nerve-roots on the right side and eroding the costal articulations. Anteroposterior röntgenogram.

miscarriages. Aside from the indigestion of which he complains and the occasional belching of gas, he was in his usual health until about the beginning of December, 1913. At this time he began to have paroxysmal spells of coughing. This cough was dry and unproductive. The paroxysms used to last about ten minutes. They were evidently influenced by the posture which he assumed, for we learn that the paroxysms were worse after he had retired. According to our interpretation, the paroxysms were worse because he was lying down. That our interpretation is correct is proved by the fact that subsequently he was obliged to sit up in a chair most of the night in order to prevent these paroxysms of coughing.

This dry and unproductive cough, undoubtedly brassy in character, continued with increasing severity until the culmination of the spitting of blood on the eighteenth of January. On that day he told me that he was riding along in a cutter when he was suddenly taken with a coughing spell. He coughed until he vomited, as people often do, and raised some blood, which he spat upon the snow. It was thus that he discovered that he spat up some blood, because of the bright-red stain which showed. He does not know the amount of blood which he spat up, but from what he said I would imagine the amount was small, probably only a dram or so. Subsequently he spat up more blood and since he has been in the hospital he has had occasional paroxysms of coughing associated with the raising of very small amounts of blood. This spitting up of occasional small amounts of blood after violent coughing is very characteristic of malignant disease. It is not characteristic of aneurysm. A dry, unproductive, brassy cough usually means either an aneurysm or a mediatisnal tumor. An aneurysm when associated with hemorrhage is usually associated with a single large hemorrhage. A mediastinal tumor when associated with hemorrhage is associated with numerous small hemorrhages, if the mediastinal tumor be a sarcoma. The very fact that in this case there have been repeated small hemorrhages is *prima facie* evidence of the existence in this man's case of a mediastinal sarcoma. There is perhaps no point in his history which furnishes such conclusive evidence of the diagnosis as this one point, and its diagnostic importance cannot be too strongly insisted upon.

The *cough* was the first symptom, beginning somewhere early in December, 1913. About the end of 1913, somewhere near Christmas, *pain* began to be a symptom of some importance. He noticed it when he was lying down. Evidently the prone posture had an affect both in producing the paroxysm of coughing and in producing the attacks of pain. The pain was located in the right upper quadrant of the abdomen, and radiated both to the epigastrium in front and backward under the right scapula up into the thorax. Very important is the fact that the pain never radiated down into the right side of the body or into the right lower abdomen or into the thigh. This pain, from its position, could be due either to some thoracic, some phrenic, or some hepatic disturbance. It is not likely to be due to renal or appendix disease.

The patient was able to describe for us the pain. He said that at first it was dull and that after a time it became shooting-like, especially in his back. He was intelligent enough to be able to assure us that it was never cramp-like. By this we are to understand that it was not associated with contraction of any circular muscle-fibers about any viscus. The fact that the pain was not colicky is rather against the suspicion that it was due to any disturbance in the gall-bladder or common duct. It is also against the idea that it was due to any ureteral disturbance. It is also against the idea that it was due to any intestinal contraction. The fact that the pain was not cramp-like again refers us to the thorax or to the diaphragm for an explanation of the pain.

A point of considerable importance which the intern got in taking the history is that the pain was increased when he raised his right arm. Such an increase of pain in mediastinal tumor and in aneurysm has been frequently noted in the right arm. There are numerous cases in medical literature where this has served as a diagnostic point in coming to a conclusion as to the existence of mediastinal disease.

Let us summarize these symptoms up to the present point in another manner. You will notice that the onset of this condition apparently began with a phrenic irritation. You will notice that at the beginning of December he had unproductive coughing spells, irritative phenomena referable to the diaphragm or the pleura. A great deal of the coughing which we meet with is really pleuritic or diaphragmatic coughing, being reflex in origin. Indeed, I believe that reflex pleuritic cough is quite as common as reflex bronchial cough. He began having these paroxysms of phrenic irritation early in December. The next step in the phrenic irritation was the production of pain in the right upper quadrant of the abdomen. I interpret this pain as due to a diaphragmatic pleurisy on the upper surface of the diaphragm, because this pain radiated around the back up beneath the right scapula into the right chest. Moreover, this pain subsequently was associated with exacerbations on raising the right arm, probably because of a phrenic nerve neuralgia and reflex brachial neuralgia.

There is another very interesting physical finding in his case which is not mentioned in the intern's history. He has a *dilatation of his right pupil;* that is, his right pupil is larger than his left pupil. The first question is whether the right pupil is dilated or the left pupil contracted. The mechanism of pupillary activity is quite simple. The third nerve contracts the pupil, a fact which can easily be remembered by recalling that atropin, by paralyzing the third nerve, permits the pupil to dilate. The sympathetic nerve is the nerve which actively dilates the pupil when the third nerve is inactive. In the case of this patient the dilated pupil must be looked upon as an active irritative phenomenon affecting the sympathetic nerve. I think that this irritative sympathetic phenomenon is associated with disturbance of the sympathetic through its connections with the phrenic supply.

Bearing in mind that the first symptom was cough, that the second symptom was pain, we are not surprised to learn that the third symptom was *tenderness*. This appeared in the right upper quadrant of the abdomen, beneath the right ribs, and extended around the spine to the back. This tenderness was constant.

On examining the patient recently we found ample explanation for this tenderness, in that pressure upon the fifth, sixth, and seventh ribs at the point of articulation with the dorsal spine produced very considerable pain. Evidently the pain in this region is intercostal in type as well as phrenic, and is probably due to a disturbance affecting the articulation of these ribs with the backbone, and giving us a hint that the malady from which he is suffering is in some way connected with the backbone and the articulations of these right ribs with the spinal column. We might easily infer an osteosarcoma originating from the bodies of the dorsal vertebræ at their articulations with the sixth, seventh, and eighth ribs.

On February 23 we learn that an operation was done by his home physicians because they believed that he was suffering from gall-stones. This diagnosis was based upon the fact that there was pain beneath the right rib, that there was tenderness over the gall-bladder on perpendicular percussion, and that there was jaundice. These symptoms are not sufficient for the diagnosis of gall-stones. There were no attacks of fever or chills or sweats. The jaundice is open to a good deal of question. According to the patient and the patient's family, he was not jaundiced. The physician was the only one who noticed the jaundice. In favor of the patient's statement is the fact that clay-colored stools were never present. Of course, jaundice is not essential to a diagnosis of gall-stones since it appears in only 50 per cent. of the cases. Still it was evidently used as a diagnostic point in this man's case, though not associated with clay-colored stools. It is, therefore, not to be wondered at that the operation was unsuccessful.

On physical examination the patient shows a good deal of pathology in his right chest. There is dulness in the right chest about the sixth rib posteriorly, running down to about the ninth rib. In front the signs are less pronounced. Very close to the sternum in front there is some hyperresonance, which is apt to be present when lung tissue is compressed. The breath-sounds are reduced over this area in front, also over the area posteriorly. Râles are present over the posterior portion of the thorax in the dull area, and seem to be due to disturbance in the lung tissue.

On account of this area of dulness associated with râles and evidences of compression of the adjacent lung tissue suspicion of a neoplasm in the mediastinum was entertained. An *x*-ray was taken and the *x*-ray shows here. Although the umbilicus is not marked in this plate, it would be at this point [indicating]. Here is the diaphragm. Here is the area of greatest dulness, which is for the most part located at the angle between the right upper surface of the diaphragm and the spinal column. You will notice that the outline is not sharply demarcated, as it should be in a case of aneurysm. At this point the lung tissue is perfectly normal. On the left side the lung seems entirely normal. On the right side there is at the lower part of the lung a little biting out, and a mass, which you will see. You will notice that the upper part of the diaphragm is not sharply demarcated along the sternum. You will also notice that this tumor mass extends from the fifth rib to the eighth rib.

The first question is, is the tumor associated with the heart or with an aneurysm of the aorta? That it is not associated with the heart is perfectly clear by the examination of the latter organ, which by auscultation is found to be perfectly normal. Moreover, there are no variations from normal on the part of the pulse. The argument against its being an aneurysm is the fact that the outline, though fairly sharp, is not perfectly so, especially along the upper edge. In support of its being an aneurysm, you can count the pulsations of the heart by observing the movements of the right nipple and also by observing the wall of the axilla. On observing these facts more carefully it will, however, be noted that these movements are communicated movements and are not due to any expansile pulsations situated in the right chest. Moreover, there is no systolic murmur to be found. With aneurysm you would expect in more than half of the cases to find a systolic murmur. In very many cases of aneurysm diastolic shock is present, but in this case no diastolic shock can be palpated. You may reason also that this thing is not an aneurysm because there is no aortic insufficiency and because there is no specific history.

Disease of the esophagus is not likely because there is no history of difficulty in swallowing. In the case of a tumor mass as large as the mass present in this case there ought to be much more in the way of symptomatology if it originated from the esophagus.

So far as I can see, there is no possibility that this mass is secondary to a process beneath the diaphragm. There is no history of duodenal or gastric ulcer or perforation of either. There is no history of appendicitis to account for the possibility of a subphrenic abscess with a breaking through into the thorax. There is no history of any infection anywhere in the portal circulation to account for the existence of a subphrenic abscess. There is no history of dysentery or amebic infection.

As a matter of fact, this man's disease originated as a pleuritic affair. It started with a cough which was increased when he was lying down. Subsequently he had a severe coughing spell which led to the spitting up of small amounts of blood, which led strongly to the suggestion of the presence of a malignant disease, particularly a mediastinal sarcoma. I think we have the key to the diagnosis in the blood-spitting. A coughing spell and the spitting of blood in small amounts are not suggestive of an aneurysm or of pulmonary tuberculosis. When we take into consideration also his very marked loss of weight—40 pounds in a few weeks' time—we feel certain that the etiologic factor must be a malignancy. Such a loss of weight would hardly occur except in a very intense purulent process of the lung, and this man is spitting up no purulent sputum. It is true that he shows a slight amount of temperature, occasionally reaching 100° F. He also shows a mild degree of leukocytosis,—11,600,—which, however, is quite in accordance with the diagnosis of sarcoma. In the differential count there is no lymphocytosis; it shows a fairly normal state of affairs. The mass cannot be due to an actinomycosis, because there are no sulphur-yellow granules in the sputum, though blood is present. In order to determine whether there was any pus which had not yet made its way out into the bronchi a needle was put into his back the day before yesterday by Dr. Murphy. The needling, however, was negative.

Mediastinal sarcoma is ordinarily associated with dulness in the thorax, lung compression, and paroxyms of coughing, which is unproductive, dry, and brassy in sound. It is not so apt to be associated with pain as an aneurysm, but it not infrequently occurs. Blood-spitting is not uncommon when the sarcoma invades the lung tissue. The blood-spitting is unlike the blood-spitting in tuberculosis in that it is not associated with pus. During the past winter we had in Mercy Hospital a similar case with a similar history of blood-spitting over quite a period of time, the case terminating fatally about Christmas.

The differential diagnosis is always between an aneurysm and a mediastinal tumor. In this man's case the great loss of weight, the absence of cardiac signs, the absence of luetic infection, the absence of tracheal tug, the extreme and sudden loss of weight, and the cachexia, which is so rapidly developing, and the evidence from the *x*-ray plates, are all considerably against the diagnosis of aneurysm. In fact, there is in this case only one diagnosis which is possible, and that is a sarcoma of the mediastinum.

COMMENTS BY DR. MURPHY

Visiting Doctor (April 30, 1914): How do you account for the adhesions in the upper abdomen which were found when the abdomen was opened?

Dr. Murphy: Such adhesions are not uncommon. We had a case here yesterday where the patient had just such adhesions, but never had complained of any trouble in the upper abdomen; and we have had numerous other similar cases. Ordinarily, unless the adhesions produce traction on the gall-bladder or constriction of the cystic duct, they are without particular significance so far as the biliary tract is concerned. The history of this man does not indicate that either adhesions or gall-stones caused the pains of which he complains so bitterly.

(Examines the patient.)

The respiratory sounds are diminished over the right lower chest, more behind than in front. A similar finding is commonly present on the first day of a lobar pneumonia when the alveoli are about half full of exudate. But the perfect tonus of this man's heart, the absence of any accentuation of the pulmonic second, and the complete absence of any trace of cyanosis, show that there is no pneumonia there. The diminution of the respiratory sounds over a pulmonary area, when accompanied by hyperresonance (Skodaic) on percussion, indicates a focus of relaxed lung tissue—atelectasis. This condition may be due not only to pneumonia, but to any pathologic process which produces pressure on the lung. If we could look this patient over with a fluoroscope, we might have some more light thrown on the nature of this round area of increased density which the *x*-ray plate shows us in the right lower chest. There is a slight lateral deviation in the spine, the convexity to the left side. This is a fixed deviation and lies on a level with the tumor seen in the *x*-ray plate. [Addressing the patient.] How much pain have you in the side? Show me just where it is. [The patient points.] The cutaneous area which the patient indicates as the site of his pain corresponds exactly with the distribution of the intercostal nerves lying on a level with the tumor. It is quite possible that they may be nerve-root pains. What kind of a pain was it?

PATIENT: At first it was a full pain (a distressing sense of fulness); then it became more severe.

DR. MURPHY: You never had to have a hypodermic because of the severity of the pain?

PATIENT: No.

DR. MURPHY: All the diagnostic points which we have elicited would fit in with a sarcoma occurring in that position. There is, first, the circumscribed area of Skodaic resonance to the right of the vertebral column, which we elicited before the *x*-ray plate was made. You see its location corresponds exactly with the mass seen in the *x*-ray plate. Then there are the diminished respiratory sounds over this same area. Then there are the spitting-up of blood, the nerve-root pains, and the deviation of the spinal column. Finally, our diagnosis of an intrathoracic tumor compressing the lung and likewise involving the spinal column is confirmed by the radiogram. The outline of the lower right costal arch is slightly bulged outward and the ribs may be somewhat eroded, otherwise it would not hurt him so much to press on his costal cartilages. That this man has an intrathoracic neoplasm is certain, and that this neoplasm is the cause of the symptoms on account of which his gall-bladder was opened and for which he seeks relief from us is equally certain. Not infrequently cases of herpes zoster, which have prodromal radiating pains very closely similar to those of this patient, are operated under the diagnosis of acute cholecystitis or acute appendicitis. Such errors are made not only during the prodromal, preëruptive stage of the disease, but also even in the presence of a florid eruption. Why? Perhaps because patients, particularly those with abdominal complaints, are examined too often in the prone position and doctors forget that these patients have backs, where zoster lesions are most frequently found. Just what is the nature of the tumor is far from certain, however. We should like to have a fluoroscopic examination made of this chest, because the fluoroscope will tell us whether or not this is a pulsating tumor. It it does not pulsate, we can rule out aneurysm with a fair degree of certainty. Of course, a very vascular tumor might also pulsate; so, in case we find pulsation present, we shall still remain somewhat in doubt. In general, however, when a thoracic aneurysm produces hemoptysis it is very close to rupture, whereas a malignant tumor of the lung may produce hemoptysis relatively early in its course. Unless some new and unexpected developments occur in this case in the future, we shall consider it in all probability a sarcoma, going out most likely from the spinal column. Without the x-ray picture we might have been inclined to regard this case as a beginning spondylitis deformans because of the neuralgic pains, the slight lateral deviation and fixation of the spinal column, and the radiating pains on fist percussion. Metastatic spondylitis is a fairly frequent condition in the locality of Chicago; it is almost equally frequently overlooked by the surgeon; and the number of these cases that are operated for chronic appendicitis, cholelithiasis, gastric and duodenal ulcer, is not small. There is often a good deal of excuse for the error. The signs and symptoms are often slight and misleading, and the x-ray plates, to detect with certainty the early cases, must be made by an expert. If at the present stage of this patient's disease we are having so much trouble trying to find out what is the matter with him, how much more difficult it must be to diagnose such a case in the early days of his illness? We have no criticism to make of the mistaken diagnosis of the surgeon who operated him for a gall-bladder lesion.

A metastatic spondylitis is one of the things that must be looked for in every case of beginning or advanced fixation of the spine. Every fall and winter, from November to March, we get a group of these spondylitis cases. They are primarily metastatic infections of the spine, the intervertebral articulations being involved. They occur in the farming districts at the season of the year when the people are husking corn, digging potatoes, carrying grain, or doing other heavy work in the cold or wet. They work in stooped positions in the fields in damp weather, strain their backs, and also catch cold. The microörganisms from the throat or nose infection enter the blood-stream and are arrested in the neighborhood of the intervertebral articulations, producing an intervertebral arthritis. In consequence of these frequently recurring or chronic arthritides, bony "lipping" of the joint edges gradually occurs, and ends in complete bony fixation of the spine. It is difficult early to differentiate such an acute spondylitis from an appendicitis or from a gall-bladder infection; but the fixation of the spine is practically always present in some degree in these cases, and particularly characteristic of this type of fixation is the fact that the spine is fixed over a considerable area, which may, and frequently does, include the entire column. It is a diffuse, not a sharply limited, focal process, and when it leads to the production of spinal deformity that deformity takes the form of a gradual curve. There is never any such angular deformity in these cases as is seen in focal processes, such as tuberculosis or metastatic carcinoma.

[NOTE.—The patient left the hospital on May 23, 1914, his condition remaining practically unchanged except that the pain was diminished somewhat by the *x*-ray treatments, given about every third day, using a rather hard tube.

He continued frequently to cough up blood during his stay in the hospital, and this condition appeared to become worse as time passed. He gradually became considerably worse after returning home, and he died early in September, 1914.—Ed.]

A TALK ON THE DIAGNOSIS OF MENINGITIS AND THE DIFFERENTIATION OF ITS VARIETIES BY DR. CHARLES L. MIX, ILLUSTRATED BY TWO CASES

Dr. Mix (May 8, 1914): The first patient I wish to show entered the hospital April 30, 1914, in the service of another physician. The patient is an Italian, a young boy of about nineteen, utterly unable to understand or to speak English. We know nothing about his medical history except what we have been able to learn from friends who have called to see him. We understand that he was recently taken sick with some sort of fever, that he had headache and vomiting, and that he was unable to do any more work on the railroad which employed him. He was, therefore, brought to the hospital and placed in a ward. Examination of the blood made May 1st, showed that he had 7600 leukocytes. A Widal reaction made the same day proved negative. An examination of his blood made again on the fifth of May showed 14,000 leukocytes. The urine showed no albumin, no sugar, an occasional hyaline cast, a few white blood-cells, and amorphous urates.

You will observe that the patient is lying with his feet drawn up, so that the thighs are flexed upon the trunk and the calves upon the thighs. You will notice that if his legs are straightened that he shortly brings them back to the contracted state, evidently because this posture affords the greatest relief. You will notice that there is no retraction of the head.

I wish first to call your attention to the history sheet of this patient. He entered at 8 p. m. on the thirtieth of April, coming in an ambulance. At the time of his admission his pulse was 80, his temperature 98.6° F., his respiration 18, a set of findings which apparently indicated no acute illness. The nurse records that at midnight his pulse was 66, his temperature 99.2° F., his respiration 26. He was very restless, and about 1 o'clock in the morning vomited some fluid of a greenish color. At 3 o'clock he was sleeping. At 4 in the morning he passed 270 c.c. of urine. The nurse records that he had a poor night and that he slept but four hours. At 6 o'clock in the morning his pulse was 51, his temperature 98° F., his respiration 18. At 8 o'clock his pulse was 70, his temperature 98.4° F., his respiration 20. At 10 o'clock he again vomited. At 3 P. M. his pulse was 60, his temperature 98.6° F., his respiration 20. He again vomited. At 6 P.M. his pulse was 62, his temperature 98.4° F., his respiration 18. At 10 P. M. his pulse was 52, his temperature 98.4° F., his respiration 22. At midnight his pulse was 50, his temperature 99.4° F., his respiration 22. I have given a summary of this first day in the hospital because upon that day, by reason of this record, we had no difficulty in making our diagnosis. We then had sufficient data, and sufficient data have already been given to you to make a tentative diagnosis. There are in this case four very important elements—headache, vomiting, a very slow pulse, and some slight fever.

Date	Pulse	Temperature	Respiration
February 25, 3 A. M.	88	101.2° F.	22
6 A. M.	86	100.2° F.	22
9 A. M.	60	99.2° F.	20
12 M.	74	99.6° F.	20
3 P. M.	56	99.2° F.	20
6 P. M.	64	98.6° F.	20
February 26, 3 A. M.	84	100.0° F.	22
6 A. M.	88	100.2° F.	24
9 A. M.	66	100.6° F.	22
12 M.	64	100.0° F.	20
3 P. M.	64	99.8° F.	22
6 P. M.	88	101.0° F.	24
9 P. M.	80	99.0° F.	22

Before saying anything further about this present case, I wish to call your attention to that of another patient who entered the hospital on the twenty-fourth day of February, and who died on the twelfth of March. This man when he entered was apparently in excellent condition. He entered at 6 P. M. on the twenty-fourth of February, walking into the ward complaining merely of pain in his head and of vomiting. He also had pain in his back, extending around to his abdomen. At 6 P. M. his pulse was 80, his temperature 101.8° F., his respiration 24. At 9 P.M. his pulse was 86, his temperature 102.6° F., his respiration 24. At midnight his pulse was 88, his temperature 101.4° F., his respiration 24. The following table shows his pulse and temperature rates during the next two days.

Why have I taken the pains to transcribe this record upon the blackboard? Simply because this record carries with it the diagnosis. There is only one acute condition which can cause such a peculiar relationship between pulse-rate and temperature, and such a peculiar oscillation of pulse-rate and temperature, and that disease is meningitis, whether of the tuberculous, of the epidemic, or of the pneumococcic variety. In the early stage of the disease the pressure of the fluid exudate is considerable. There is a marked irritation of the pneumogastric inhibitory fibers, with the result that the pulse-rate is remarkably slow. In the case of this man whose temperature record has been transcribed, only four symptoms were ever presented. These are: slow pulse, variable temperature, headache, and vomiting. Upon these four signs and symptoms the diagnosis of meningitis was tentatively made and a lumbar puncture done. The result of the lumbar puncture was that a perfectly clear fluid was obtained, which on microscopic examination showed no bacteria and hardly more than the ordinary number of lymphocytes after vigorous centrifuging. The cerebrospinal fluid, however, was found to be under tension when the lumbar puncture was made. I had a guinea-pig injected with this cerebrospinal fluid on the third of March, 1914, and the guinea-pig died on the fourteenth day of April, just forty-two days after the injection. The post-mortem on the guinea-pig made by Professor F. Robert Zeit, of Northwestern University Medical School, showed enlarged caseous inguinal glands and a miliary tuberculosis of the spleen. Tubercle bacilli were found in the smears from both the inguinal glands and the spleen. Meanwhile the patient himself had died on the twelfth day of March, showing no further symptomatology than the development of semi-coma and then of coma. After the lumbar puncture was made the man made quite an improvement, so that one of the visiting physicians jokingly told me that my tuberculous meningitis case was getting well. I told him to wait and see.

Whenever you run upon a case without labial cold sores, with an insidious onset, with the presence of headache of a severe and continuous type, with vomiting not to be explained on the basis of any disturbance of the alimentary canal, with a pulse in the fifties, and a temperature above normal, it is always your duty to bear constantly in mind the possibility of tuberculous meningitis. It is always your duty to do a lumbar puncture in these suspected cases, and, furthermore, to inoculate a guinea-pig with one or two cubic centimeters of the cerebrospinal fluid.

Returning now to the patient whom we are at present considering, I would like to call your attention to his temperature range on the second, third, and fourth of May.

Date	Pulse	Temperature	Respiration
May 2, 6 A. M.	54	98.2° F.	22
9 A. M.	50	97.8° F.	22
12 M.	54	98.0° F.	20
3 P. M.	54	100.2° F.	20
6 P. M.	60	98.6° F.	20
9 P. M.	54	99.0° F.	20
12 P. M.	56	99.0° F.	22
May 3, 3 A. M.	Sleeping	..	..
6 A. M.	46	98.6° F.	20
9 A. M.	54	97.8° F.	20
12 M.	52	97.6° F.	20
3 P. M.	60	99.6° F.	24
6 P. M.	52	99.2° F.	26
9 P. M.	80	101.0° F.	26
12 P. M.	66	99.0° F.	22
May 4, 6 A. M.	78	100.4° F.	20
9 A. M.	68	99.6° F.	20
12 M.	60	98.8° F.	22
3 P. M.	72	100.0° F.	22
6 P. M.	68	99.6° F.	20
9 P. M.	72	99.2° F.	28

You will notice the great similarity between this set of pulse-rates and temperatures and the case of the man whom I just mentioned, whose death, together with that of the guinea-pig, proved the presence of tuberculous meningitis. So closely similar were these history sheets that the intern recognized at once that the cases were identical in type.

On the second of May the young Italian slept a great deal of the time and in the late afternoon complained of soreness and aching in his neck and shoulders. In the night he tried to get out of his bed, being apparently somewhat irrational. During most of the forenoon of the third of May he slept. About noontime he began picking at his face and nose in an apparently aimless manner, his temperature at this time being one degree subnormal. It was on this day that I was first called to see him and did the lumbar puncture At that time he seemed somewhat irrational, but, of course, his inability to speak English and my inability to speak Italian made conversation rather difficult. The nurse reports that during the night he was again very restless. On the morning of the fourth the nurse noted that he was irrational all the forenoon, and in the afternoon apparently did not recognize anybody. On the fifth of May for the first time he began having involuntaries. During this day he began to moan in his sleep and his limbs twitched. The nurse reports that during the forenoon of the fifth he was apparently unconscious. He remained unconscious, or at least semi-unconscious, with involuntaries during the day of the sixth, until late at night, when he tried to talk a little.

On the fifth of May a marked change took place in his pulse-rate. At 3 A. M. it was 78; at 12 M. it was 78; at 3 P. M. 96, at 9 P. M. 110. On the sixth of May it was 110 at 1 A. M., 128 at 6 A. M., 118 at 8 A. M., 112 at noon, and 120 the rest of the day. On the seventh his pulse varied between 104 and 150. This extreme variation took place within three hours' time. At 3 A. M. his pulse was 112; at 6 A. M., 148; at 9 A. M., 126; at noon, 124; at 3 P. M. 150, with a temperature of 100.6° F.; at 6 P. M. 104, with a temperature of 100° F.; at 9 P. M. 128, with a temperature of 99.6° F.; at midnight 138, with a temperature of 99.8° F. This morning his pulse is 112 and his temperature 98.8° F.

Yesterday morning early he was pulling at the covers without apparently knowing what he was doing, but at 9 o'clock he became conscious and tried to talk and moved his hands and legs about with some show of reason. He was conscious all yesterday afternoon, though during the night he talked and moaned a good deal. This morning he has been quite rational and is rational at the present time. Notwithstanding the fact that he is at the present rational, he had this morning involuntary passages of urine as usual.

Physical examination in this young man's case shows, besides this remarkable relationship between pulse and temperature, a few facts which are rather characteristic of meningitis. First of all he assumes a posture in bed which is assumed by most patients suffering from this disease. His neck and head are not retracted, it is true, neither are they stiff, as you can see, when I attempt to lift his head, but you will notice, as I said before, that his feet and his legs are drawn up, his thighs being flexed on the abdomen and the calves on the thighs. You will notice also that when I touch him he is apparently hurt by the touch. That is to say, he resents the touch and moans when his limbs are changed in their position.

You will notice further that Kernig's sign is present; that is to say, when his thighs are flexed upon his abdomen, the legs cannot be forcibly extended. This is because of the contracture which is taking place in his hamstring muscles. This contraction is an evidence of cerebrospinal meningitis. It is by no means confined to the semimembranosus, semitendinosus, and biceps femoris muscles. It is found all over the body. Kernig's sign is of importance not so much in itself as in proving the presence of a hypertonic state of the muscles of the body. The muscles of the trunk share in this hypertonic state and the retraction of the head and neck, so commonly a picture in meningitis, is to the muscles of the back what Kernig's sign is to the hamstring muscles, namely, a sign of increased muscle tonus. The arms also show this same phenomenon. You will notice that the forearms are flexed upon the arm. When I try to straighten them, the patient moans slightly and is apparently caused some pain. The reason is that we have a hypertonic state in the biceps cubiti which must be overcome if the arm is to be extended; and overcoming this hypertonus causes pain. Sometimes hypertonus of the erector spinæ muscles causes in some cases a condition of opisthotonos of mild degree.

You will notice that the patient also shows marked sensitiveness of the skin. Leube long since called attention to the hyperesthesia of the skin and muscles in meningitis. Personally I regard it as one of the most characteristic features of the disease. To me Kernig's sign is no more valuable a diagnostic point than the hypersensitiveness which is present in the patient's body during the progress of the meningitis.

Turning now to another set of phenomena which makes the diagnosis of these cases most certain, we will look at the boy's pupils. You will notice that the left pupil is larger than the right. This inequality is very variable, as is also the size of the pupils. In the early stages of meningitis the pupils are very apt to be contracted. Later on they become dilated, but usually unequally, so that one pupil may be mydriatic while the other is normal in diameter. The variability of the pupils rather than the mere presence of inequality is the important fact. You may find one pupil widely dilated in one hour and perhaps the next hour find it of normal size.

In these cases the muscles of the eyeballs are frequently involved, and a very common finding is ptosis of one or the other of the levator palpebræ superioris muscles. Frequently there is a strabismus, quite as often due to the paresis of the internal rectus as of the external rectus. We find, therefore, that the eyeball may sometimes turn in or sometimes may turn out. In this boy's case there has been previously a paresis of both of the internal recti muscles, though there is nothing to be seen at the present time.

Summarizing his symptomatology, we have the remarkable pulse and temperature relationship—a very slow pulse in the early stages, the presence of severe headache with vomiting; the presence of pupillary inequality and variability in the size, with transitory internal strabismus. We need no further data to make a diagnosis of meningitis, but we do need further data in order to determine the *type* of meningitis present—whether it is of the tuberculous or epidemic variety. Is it a streptococcic meningitis? Is it one of those rarer forms due to the typhoid fever bacillus, the influenza bacillus, the staphylococcus, or the pneumococcus? These questions can be answered very quickly if we have recourse to a lumbar puncture. It goes without saying that the first time we saw this patient we had recourse to a lumbar puncture. We obtained a fluid which was apparently perfectly clear to the naked eye, but which on centrifuging showed more than the very few lymphocytes obtained in centrifuging normal cerebrospinal fluid. We have injected into a guinea-pig one cubic centimeter of the cerebrospinal fluid. This we did on May 4th. We expect the guinea-pig to die of tuberculosis.* We expect him to die of tuberculosis because the cerebrospinal fluid was clear. Had any of the germs which have been above mentioned been the cause of the meningitis we should have found numerous polymorphonuclear cells in the fluid, and a test-tube of it would have given the appearance of ground-glass or an almost opaque fluid, varying from a haze to a complete opacity. On one occasion on doing a lumbar puncture I actually withdrew pus from the cerebrospinal canal due to a staphylococcic meningitis, from which the patient died on the following day, the case up to that time having been regarded as typhoid fever, though no Widal had been present.

The fluid in the case of this young man came out with a good deal of rapidity, indicating quite a bit of tension. Often after such a lumbar puncture the patient is better, but no improvement followed in the case of this individual.

Another reason why we say this case is a tuberculous meningitis rather than a meningitis caused by some other bacterium is because of the absence of herpes and of signs of infection of any sort elsewhere in the body. Many cases of meningitis have their etiology indicated by disease elsewhere, so that it is a very simple thing to arrive at a diagnosis. Thus in typhoid fever the signs of meningitis confirmed by a lumbar puncture point clearly to the typhoid bacillus as the cause. This cause, however, is extremely rare, Ohlmacher having reported originally a series of 12 cases only. On the other hand, if the individual is suffering from abscess of the middle ear due to some form of streptococcus, one has no difficulty in diagnosing the meningitis when it develops and in being positive as to the germ causing the meningitis.

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* The guinea-pig was injected on May 4th with one cubic centimeter of cerebrospinal fluid. This guinea-pig died on June 12, 1914, thirty-nine days later. Most guinea-pigs die on the fortieth day when injected with fluid which contains tubercle bacilli. This guinea-pig died on the thirty-ninth day, thus proving the rule. The postmortem on the guinea-pig was performed by Professor F. Robert Zeit, Professor of Pathology, Northwestern University Medical School. It showed: 1. Tuberculous peritonitis. 2. Miliary tuberculosis of spleen and liver. 3. Tuberculous lymphadenitis of the inguinal and mediastinal glands.

This is a case of meningitis which developed out of a clear sky and which must be one of four sorts—either tuberculous, epidemic, influenzal, or pneumococcic. Tuberculous meningitis is insidious in its onset, is usually associated with slow pulse, and is much more apt to have convulsions than the other three forms. The pneumococcic variety is almost invariably associated with severe cold sores. The influenzal type occurs only during epidemics of influenza, and is usually associated with evidences of influenza of an incipient type. The epidemic variety, like the pneumococcic, is usually associated with cold sores, so that the facial appearance of the two is very much the same. Indeed, so closely may these two types resemble each other that the differentiation may be impossible except by the examination of the cerebrospinal fluid or by a blood culture.

The leukocyte count helps very much in the differentiation of the tuberculous forms from the streptococcic, epidemic, influenzal, and pneumococcic varieties. All four of the latter have very high leukocyte counts, especially the epidemic form. The leukocyte count in the tuberculous form is either normal or slightly above normal, being in the case of this young Italian 7600 on the first of May and 14,200 on the fifth. In the case of the man whose temperatures I gave you at first and who died from a tuberculous meningitis on the twelfth of March the blood count on the twenty-fifth of February was 8600 leukocytes, and on the twenty-eighth of February 8000 leukocytes.

PNEUMOCOCCUS MENINGITIS

The next patient is a married woman who entered the hospital with the following history:

(Intern reads history.)

Patient, female, aged twenty-three, married, was brought to the hospital Friday evening, May 1, 1914. Her past history is negative. Her mother died three years ago from an injury resulting from a fall. Father is living and well. She has six sisters and three brothers living and well. Her husband is living and well.

Menstruation began at the age of fifteen, regular, twenty-eight-day type, three or four days' duration, four napkins a day, with little pain. She has had no children and no miscarriages. Her last period was in September, 1913, and she is seven months' pregnant.

Her present illness dates back to April 25th. Her husband states that when he returned from work the patient complained of a severe frontal headache, which had come on suddenly that afternoon. On Sunday morning, April 26th, about 9 A. M., she vomited for the first time. The vomiting was projectile in type and had no relation to eating. She was not nauseated. A doctor was called who said she had the grip, for which he gave her some medicine. The headache continued on the twenty-sixth, twenty-seventh, twenty-eighth, and twenty-ninth of April. She also vomited many times during these four days, and it was always projectile in type. On Wednesday morning the headache became occipital and was still intense in character. She did not vomit after Thursday morning, but the headache continued.

She has had no cough and no pain, excepting the headache. She had a slight backache in the lumbar region on Sunday morning, but this lasted only a short time. On Thursday night she felt much better and slept well. On Friday morning her husband says that she had a temperature for the first time, and on that afternoon became unconscious.

DR. MIX: You will notice in the history which has just been read that the patient's trouble began rather abruptly with headache on the twenty-fifth of April. Sunday morning, April 26th, a few hours after the onset of the headache, she vomited and the vomiting was projectile in type. The headache and vomiting continued until the time when she entered the hospital; and the vomiting, you will notice, was always projectile. Were no other statements made one might suppose that the patient was suffering from a cerebral tumor and one might, with every reason in the world, conclude that she was at least suffering from an increased intracranial pressure from some cause, inflammatory or neoplasmic. The history states that there was no cough or pain. There was, however, a slight backache on Sunday morning in the lower part of the spine. Her husband notes that the temperature appeared for the first time on Friday morning, that is, almost a week after she was taken ill. We believe this to be a mistaken observation. Probably by Friday the fever became so high that it for the first time impressed him. On the afternoon of Friday the husband states that his wife became unconscious.

Let me first call your attention to the history sheet which this patient shows. The leukocyte count made on the second of May showed 28,600; a second, made on the fourth, showed 21,800. The patient entered the hospital at 10 P. M. on the night of the first of May, coming in an ambulance, her pulse being 104, her temperature 100° F., her respiration 28. During the night she had involuntaries. There was severe hiccup, lasting fifteen minutes, and the patient tried repeatedly to get out of bed. She was delirious or semi-comatose all night. The following morning, on the second of May, she dozed and slept some and was quite rational at times, her pulse falling from 112 to 100, her temperature being 98.4° F., the forepart of the day, but rising to 102° F. at midnight. At 9 P. M. of the second of May her pulse was 102; at midnight, 130. At 3 A. M. on the third her pulse was 160; at 6 A. M. it was still 160; at 9 A. M. it dropped back to 104. During the third she had some slight nose-bleeding in the afternoon, was restless all the day, and had involuntaries. On the fourth of May her pulse was remarkably better, being 102 at

3 A. M.; 106 at 6 A. M.; 116 at 9 A. M.; 98 at noon; 100 at 3 P. M.; 106 at 6 P. M.; 104 at 9 P. M.; 100 at midnight. During that day she rested fairly quietly and spent a good night. The following day, the fifth of May, her respiration varied from 22 to 34, and her pulse from 90 to 130; her temperature from 98° to 103.4° F. On the sixth of May her pulse varied from 88 to 116, her temperature from 98.4° to 102.6° F. She was somewhat restless, but on the whole passed a fairly good day. Yesterday, the seventh, her pulse range was from 100 to 120. The nurse notes that she had a fair day.

This morning, at 3 A. M., her pulse was 96, her temperature 99° F., and her respiration 26. At this time she had a slight chill which lasted four minutes. A short time after that there was a gush of blood from the vagina and she was delivered of a three-pound baby at 4.20 this morning. (The baby lived only thirty-six hours.) The placenta was delivered intact at 4.30. This morning, at 9 A. M., her pulse was 104, temperature 97° F., respiration 24. She has slept quietly and seems in fairly good condition.

I wish to call your attention to the symptoms which this patient shows. First of all, you will note upon her lips a large number of cold sores. Indeed, these cold sores carry with them the diagnosis, and when I first saw this patient, noting the cold sores and reading the history and seeing the temperature chart, I could not help but feel that a pneumococcic infection was present. To be sure, an epidemic cerebrospinal meningitis case might present the same sort of a picture, but the cold sores were so very like the cold sores of an ordinary pneumococcic case that when I first glanced at the patient before looking at her history I mentally wondered whether she was not suffering from pneumonia. The first thing I did was to examine her chest and to prove to my own satisfaction that her lungs were perfectly normal. Indeed, her respiratory rate during most of the days that she has been in the hospital varied only between 22 and 34. You will note this morning on examination of the lungs that there are no areas of consolidation either in front or in the back.

On examination of the heart there was to be found when I first looked at the patient a systolic murmur from a mitral insufficiency. We have learned from her husband that this is an old lesion that she has had for a long time, and which dated from a previous rheumatic fever. The murmur which she has apparently has in no way been disturbed by her present illness. It is neither louder nor fainter than it has been at any time, and the heart is evidently in fair condition.

Examination of the blood, which was made, showed that the leukocytosis was made up largely of polymorphonuclear cells. In the tuberculous variety the leukocyte count is usually normal or slightly above normal, seldom if ever rising as high as 20,000. In pneumococcic meningitis and in the epidemic, influenzal, and streptococcic varieties the blood count is always very high, the excess of corpuscles being made up of polymorphonuclear cells. In the tuberculous cases the eosinophiles are either decreased or entirely absent.

You will note that this patient also has Kernig's sign; that there is some hypersensitiveness of the skin; some stiffness and soreness of the muscles, and the same rigidity that was present in the case of the Italian boy suffering from a tuberculous meningitis. You will note further the presence of an extreme degree of vasomotor irritability in the shape of the so-called *tâche cérébrale.* It is not the red or white streak which appears after sweeping your finger over the skin which is important. It is the vasomotor irritability which this *tâche cérébrale* shows which is the important thing. This vasomotor irritability and vasomotor paresis are of great diagnostic value in cerebrospinal meningitis.

You will note the posture of this patient in bed is similar to that taken by the Italian boy. You will notice, however, that there is more rigidity in the neck muscles than he showed.

Passing to the abdomen, you will notice that the abdomen is not scaphoid, neither was there any scaphoidism in the case of the young Italian. It has been frequently said in text-books that a scaphoid abdomen is an extremely common thing in tuberculous meningitis, but that it is not apt to be present in the other forms. I have never been able to satisfy myself as to the truth of this statement. A scaphoid abdomen ordinarily means absorption of mesenteric fat, together with excessive rigidity of the anterior abdominal wall. This excessive rigidity is merely a part of the general rigidity uniformly present. So far as I have been able to observe, the cases which survive for some time all show the scaphoid abdomen, no matter what the type of meningitis. Those living only a short time will not show the scaphoid abdomen.

This patient has had no convulsions. Usually the pneumococcic cases are entirely free from convulsions. Osler, in his text-book, states that he has never seen a convulsion in the case of pneumococcic meningitis. The convulsions are more characteristic of the tuberculous type. They also occur, of course, in the epidemic cases. They are practically unknown in the pneumococcic cases.

I wish to call attention, furthermore, to the fact that this patient vomited a good deal at the beginning. I wish further to emphasize that the vomiting was of the projectile type, indicating increased intracranial pressure, undoubtedly from the products of the meningitis. Cerebral vomiting in the cases of meningitis is usually not associated with the presence of bile. These patients take their nourishment fairly freely and yet vomit, the underlying cause of the vomiting not being due to disturbances of the stomach and intestines. When tuberculous or pneumococcic meningitis cases begin to vomit they do so until the stomach is entirely empty. If they get no more food there is vomiting usually merely of mucus and gastric juice.

Very important in the examination of these cases is the mental state. All of them show interference with consciousness. Delirium is usually early and sometimes very pronounced. It is the same type of delirium that is met with in typhoid fever. In typhoid fever there is often a condition of meningism, namely, a condition of toxicity of the cortex. The symptoms which it produces in the nerves, blood-vessels, and various muscles of the body, subsultus tendinum, are the very same symptoms which are present in cases of meningitis. The delirium is usually of the quiet type, the patient fumbling about or trying to get out of bed. Usually this delirium after a time passes over into coma with involuntaries, this coma ultimately giving place to death. Sometimes there is a severe convulsion followed by delirium, the delirium terminating in coma and the coma terminating in death. These are the usual stages of a long case of meningitis, whether of the tuberculous, epidemic, or pneumococcic variety.

Further examination of the patient now before us discloses certain defects of the cranial nerves. You will note that there is inequality of the pupils; that there is, furthermore, a ptosis of the left eyelid, and that there is an internal strabismus. The facts which we have thus far brought out in regard to the case are convincing that meningitis is present, but thus far they are not convincing as to the type of meningitis. The cold sores present are certainly suggestive of the pneumococcus. The track of the pneumococcus is shown by the cold sores which it leaves behind it, and by its tracks you can often infer its presence. The blood culture which I ordered in the case of this patient at the same time that I made the lumbar puncture showed pneumococci present. The Widal, previously ordered by the intern, was negative. Of course, in the presence of a leukocyte count of 28,600, made on May 2, a Widal test was entirely unnecessary, for no case of typhoid fever ever shows a leukocyte count of 28,600. It was, however, ordered and made, and the result, of course, was negative.

I made a lumbar puncture in this case the first day I saw the patient. We obtained a cerebrospinal fluid which was quite turbid by reason of the large number of polymorphonuclear cells which it contained. On examination of this cerebrospinal fluid pneumococci were found. There were no diplococci within the cells. The pneumococci were free in the fluid. We thus have in this case two reasons for believing that the patient has a pneumococcic meningitis, namely, the positive blood culture of pneumococci and the finding of the same pneumococci in the cerebrospinal fluid after a lumbar puncture.

I wish to call attention to one peculiar point in this patient's case. On the second of May, at 9 P. M., her pulse was 102; at midnight, 130; at 3 A. M., 160; at 6 A. M. it still remained 160;

but at 9 A. M. it dropped to 104. The patient entered the hospital about seven months pregnant. During all the time of her illness the fetal heart has been clearly heard, and this morning at 4.20 she gave birth to a seven months' baby which is still living. The sudden increase of the pulse from 102 to 160 and its sudden fall to 104 is rather difficult of explanation. I believe, however, that it is due not to any disturbance of the pregnancy, because we know that no disturbance took place, but that it was rather caused by some disturbance of the cardiac inhibitory apparatus. There is in all cases of meningitis great irritability on the part of the inhibitory nerves from the vagus to the heart. In the early stages of meningitis, during the period of muscular irritability, rigidity, and hypertonus, there is also a marked irritability of the cardiac pneumogastric fibers. This is shown by the fact that the pulse-rate often runs as low as 50, as in the case of our Italian boy. I look upon this very slow pulse as being due to irritation (that is to say, stimulation) of the cardiac inhibitory center in the floor of the fourth ventricle, either because of direct pressure upon it from the increased pressure in the cerebrospinal fluid or because of irritation by the toxins of the pneumococci upon the cardiac inhibitory center. There is at any rate in the early stage a marked irritability of these cardiac fibers. The result of the increased irritation is a marked slowing of the pulse—just such a slowing as one might obtain by artificial stimulation of the cardiac inhibitory fibers of the pneumogastric. In this particular case of pneumococcic meningitis the sudden acceleration of the pulse to 160 probably marked the stage in which the irritation of that apparatus, by reason of its great excess, passed over into a stage of complete paralysis of that apparatus. The result would be that no more inhibition would take place and consequently the pulse would run the rapid and alarming rate of 160. The same thing happens previous to death in the tuberculous, pneumococcic, and epidemic cases of meningitis, and a very rapid pulse is almost invariably recorded in the last day or twelve hours of these patients' lives. The reason is that the toxicity of the blood has reached such a degree that the cardiac inhibitory apparatus is completely paralyzed;

and the paralysis is so deep that it is never recovered from and death follows within a few hours' time. Such was the case with the man with the tuberculous meningitis who died on the twelfth of March. His pulse on the eleventh was 106 at noon time; 108 at 3 P. M.; 108 at 6 P. M.; 112 at 9 P. M. At this time the patient began to be very restless, talked a good deal, and was perspiring freely. His pulse then went up to 140 at midnight; at 2 A. M. 160, and thereafter the nurse was unable to count it at all. At 8.30 A. M. his respirations ceased. We have here again the same acceleration of the pulse before death that takes place in the great majority of these cases. I believe that death in practically all the cases of cerebrospinal fever is primarily due to a paralysis of the cardiac inhibitory fibers with secondary paralysis of respiration.

Returning now to our case of pneumococcic meningitis, I am going to do a lumbar puncture and then Dr. Murphy is going to speak about the treatment of these cases. The technic of a lumbar puncture is extremely simple. You merely find the crest of the ilium and draw a line across the back, midway from one crest to the other, locating the first spinous process above this imaginary line with the finger. The skin is then painted with iodin. You then insert your needle, which should be two and three-fourths or three inches in length into the back, about one-fourth of an inch from the midline, either to the right or the left of the spinous process. The needle is pointed in a direction upward very slightly and toward the midline, at such an angle that the tip of the needle will be in the midline when it is admitted to a depth of about one and one-half to two inches. When you insert your needle you will usually feel in the majority of instances the point coming in contact with the vertebral lamina. You then manipulate your needle, pointing it a little higher upward, so that it just skips the edge of the lamina. As the needle passes into the spinal canal you can distinctly feel it going into a hole, which it does very easily. You then release your finger from the needle end and the cerebrospinal fluid usually passes out at once. It often passes out under very great tension.

The reason for doing a lumbar puncture this morning is not for diagnostic purposes at all. It is a part of the treatment of these cases. These patients show a varying degree of somnolence and coma, depending upon the varying degrees of pressure; and the degree of pressure can be controlled by occasionally depleting the cerebrospinal fluid by puncturing. Were this a case of epidemic cerebrospinal fever we would not only remove 20 to 30 cubic centimeters or more of the cerebrospinal fluid, but we would also insert into the spinal canal a dose of Flexner's serum. This being a case of pneumococcic meningitis, we merely deplete the cerebrospinal fluid without inserting anything in its place.

COMMENTS

Dr. Murphy (May 8, 1914): Dr. Mix is puncturing the spine in this case as a therapeutic measure. The excess of intracranial and spinal pressure is making the patient comatose. By carrying out this simple procedure, we believe we are treating the case from a well-grounded surgical standpoint. Infection does its mischief through the tension under which its products are held, in practically every variety of inflammation except the streptococcus infections. The streptococcus infections do not do much mischief, as a rule, through the tension under which the products of inflammation are held, because the streptococci run freely through the lymphatics, usually, are not held *in loco*, and, therefore, do not produce tense foci of pus; while staphylococci and pneumococci are held *in situ* by the resistance to their spread, and by the inflammatory coffer-damming of the tissues from blocking of the reticular and lymphatic spaces. The pus keeps on destroying tissues in proportion to the number of polymorphonuclear leukocytes which are present in it, and the tension under which it is held. In an ordinary mycotic abscess of the thigh, what does one do? One just removes the tension and the tissue destruction stops. It is not necessary to go in and clean out the abscess and scrape its walls. One removes the tension by opening the abscess, and one keeps the tension down by maintaining free drainage as healing takes place.

I said the tissues were destroyed by the necrobiotic and toxic action of the pus under tension in proportion to the number of polymorphonuclear leukocytes contained in it. Why do the polymorphonuclear leukocytes tend to destroy the tissues where they are held *in loco?* It is because of the trypsin which they contain. It is the trypsin which does the mischief through its ability to digest the proteins of which the body tissues are made. In a case of purulent meningitis the toxic and necrobiotic action of the pus is enhanced by the inelastic wall of the cranium, which cannot give and stretch like the soft tissues. The evil effects of pus under tension are exerted in double measure on the delicate and vital elements of the brain because of the absolutely unyielding character of the cranium and the relatively slight extent to which the brain itself can give way because of its inclosure in this bony box. Somewhat the same conditions obtain as with a central osteomyelitis. The center of a long bone is soft, but it has an inelastic wall about it, the cortex; consequently, destruction of the medulla progresses at an alarming rate, and extensive or complete destruction of the marrow may ensue before the cortex shows any appreciable gross involvement. The same is the case in infections of the brain, the infection early forcing its products into the circulation, rendering the adjacent brain tissue first ischemic and then necrotic. Dr. Mix is puncturing the spinal canal as a part of the treatment of the case, not merely as a diagnostic measure; and the procedure will be repeated tomorrow and the next day. He notices today a pronounced change in the character of the fluid since yesterday. It is much less purulent.

What will be gained by this patient from this procedure? Let us see. The microörganism present in the spinal fluid is the pneumococcus. Pneumonia is a self-limited disease. It produces its intoxication early and promptly, and its microörganisms are destroyed, if the patient survives, at a definite date regularly —in children, from the seventh day on; in adults in midlife, about the ninth day, as a rule; in the senile, on the eleventh or twelfth day. In the early days of bacteriology it used to be thought that the non-fatal cases of these infections got well because the invading organisms were destroyed by intoxication from the progressive increase of their own toxins in the circulating medium of their host, just as bacteria gradually die out, in the course of time, on culture-media *in vitro*. We know now that the microörganisms are destroyed in these non-fatal cases of infection by the action of antitoxins and antibodies produced by the host's body tissues and blood-cells, a radically different and far more hopeful theory than the former, for it points out to us a road to cure. In this case treatment is chiefly a matter of carrying the woman along a sufficient number of days with supportive treatment until this infection subsides, that is, is overcome by antibody formation. If one can prevent the progressive increase in intracranial and spinal pressure from developing to a fatal degree during the time the infection is active, and if other foci of pneumococcus infection do not arise elsewhere, she may get well; but we know from experience that pneumococcus meningitis has a very dubious outlook, because of the practical difficulties in meeting successfully these therapeutic indications. What we need most, and still hope for, for these cases is an antipneumococcus serum of real efficiency. There is none yet.

Pneumococci are present many times in the mouths of normal individuals. In persons who have had pneumonia once the organism is present in the mouth in 60 per cent. of the cases. In those who have had the infection twice the organism is present in the mouth in almost 100 per cent. of the cases. It is present in the mouth under normal conditions in an enormous number of cases who have never suffered from any active manifestation of pneumococcus infection. So numerous are these pneumococcus-carriers that the astonishing feature about them is that the pneumococci find entrance into their blood so rarely. What we should wonder at is that we have so little pneumonia—not so much. Although the organism often appears to act merely as a harmless saprophyte, yet when it does find entrance into the blood through the tonsils or alveolar processes, or through the pharyngeal or bronchial lymphatics, or through infection of an artery or vein, it often produces one of the most virulent types of general infection that is possible. Following the extraction of teeth and in connection with fractures of the jaw there not infrequently develops a pneumococcus infection spreading from the site of injury.

I recall one case that occurred on my service at the Presbyterian Hospital. A boy had a tooth extracted. He had a little more pain that night than he had had during the day. The next day he had a chill. Finally, on the second day after the extraction, he was brought to the hospital. On the morning of the third day a pure pneumococcus culture was grown from his blood. Then he had another chill. Then he commenced to show signs of an acutely developing endocarditis, of which Dr. Mix said this patient has no manifestations, which is proof that the infection is still held within her cerebrospinal tract. When the endocarditis was in full bloom, the boy's heart valves commenced to throw off emboli. These emboli were carried by the arterial stream to various organs in the body. First he had a gangrene of the right leg; then an infarct in the kidney with blood in the urine; then an infarct in the spleen; then in the lung; he developed a bloody expectoration from the multiple infarcts in the lung; then came a gangrene of the left arm. He lived on until, finally, one morning an embolus slipped into one of his cerebral arteries; he was dead in thirty seconds.

I cite this case merely to show how numerous and deadly the infective emboli may be and how rapidly they are sometimes thrown off in an intravascular infectious process, particularly when the infection is virulent and located on the valves of the heart. There are in the museum of Rush College the specimens of this case, obtained at autopsy. Every one of these emboli is preserved and the specimens are arranged in temporal order, showing the sequence in which they occurred. And all this beautiful pathologic collection came from the simple extraction of a tooth!

One of the other conditions in which there is great danger of a complicating pneumococcus infection is fracture of the base of the skull, and particularly that type of fracture where there is an escape of cerebrospinal fluid. Such a case, for instance, as we had here some ten days ago, runs a great risk of acquiring a fatal pneumococcus meningitis. The patient had, if you remember, a fracture of the base of the skull, with the cerebrospinal fluid—a clear fluid—discharging from his nose for many days after the injury. Finally, however, the opening closed up, and the danger of infection was over; but such is not always the outcome, the opening occasionally remaining patent and discharging for months and years. I recall a case occurring in my very early practice—a child who had a discharge of cerebrospinal fluid following a fracture of the base of the skull. The discharge persisted for six or eight years. I was conjecturing as to how to close the opening by operation when, all of a sudden, the child developed a pneumonia which went on to a rapidly fatal termination, perhaps a fortunate occurrence for the child for it was already commencing to show vicious mental symptoms with a gradually progressive epilepsy.

Dr. Mix will show you these cases again from time to time, provided they do not die. It is delightful to be able to study cases of this class worked up in so scientific a way, so that one knows just what one is doing at every step and why. Every phase of the disease has been worked out in these cases by Dr. Mix and his assistants. One also knows, unfortunately, some of the limitations of therapy in these cases. For this patient Dr. Mix is developing a vaccine, and I presume that by tonight or tomorrow morning we shall have an auto-vaccine for her, which should be another element contributing toward tissue repair, through increasing the body resistance to the infective agent. By making repeated lumbar punctures Dr. Mix will try to keep down the increased intracranial pressure, the immediately most serious symptom which this patient shows. I was very happy to have Dr. Mix take a half-hour of my clinic time to show these two cases, because they shine by mutual contrast. They are sufficiently common to make one wish to know more about them and to know it in an exact way, so that when one again has such patients come under observation he will not have the experience which I had once in my early days of medical practice. When I began practice there was a family, the patients of Dr. E. W. Lee, with whom I was then associated, living on South Morgan Street, near Sixteenth. Dr. Lee was a very able man, a splendid diagnostician, and a thorough clinician. He had doctored members of this family for a long time. One or two of the children had already died of tuberculous meningitis. One day, in Dr. Lee's absence, I was called over to examine one of the children. I looked the little patient over carefully and said, very wisely, so I thought, to the mother that the child had merely a bronchitis—nothing to worry about. Bronchitis is not at all uncommon as an initial symptom of tuberculous meningitis. After I got all through delivering my learned and, supposedly, reassuring opinion, the mother said to me, "Doctor, this is no bronchitis. It is a meningitis that this child has." I asked, "What makes you think so?" She replied, "I have had two children die of it and they both started in like that. If you think this is a bronchitis, I shall have to get another doctor." She did get another doctor, and the child died because it did have a miliary tuberculosis and a tuberculous meningitis; but she continued to keep Dr. Lee as her family doctor, in spite of his assistant's errors. He had made the correct diagnosis and prognosis in the previous meningitis cases, and they both died just as he said they would, thoroughly confirming the woman's confidence in Dr. Lee's medical infallibility. When she happened to be in the office and saw me pass through she used to say to Dr. Lee, "Every time I see that man it gives me a chill. I pity his patients who don't know him so well as I do." I hope you have all learned to make these diagnoses so correctly and promptly that people will not have a chill when they see you, but will be reminded of your infallibility, and will come to lean on you as on the Staff of Life. I was very happy, for that reason, that Dr. Mix went over the details of these cases, because diagnosis must always be worked out largely on the basis of the clinical course of the patient's illness. Case after case makes the diagnostician. One cannot become a diagnostician from reading books only. One can help one's self greatly toward medical proficiency by judicious reading, and reading is a great aid in developing one's power of medical observation; but seeing the cases is the essential thing.

[Note.—The Italian boy with the tuberculous meningitis died at 5.55 A. M. on the morning of the tenth of May. He fulfilled the usual rule as to the acceleration of the pulse-rate before death. On the ninth his pulse was 120 at 9 A. M.; 144 at noon; 148 at 3 P. M.; 160 at 6 P. M.; and 150 at 11 P. M. On the morning of the tenth the nurse was unable to count his pulse, but recorded it as 150 at 3 A. M. and 158 at 4 A. M. After that the pulse is recorded only with a question mark. The patient expired at five minutes to six, May 10th. The patient with the pneumococcic meningitis had the same kind of an ending, dying at 9.45 P. M. on the ninth of May. At noon on the ninth her pulse was 100; 1 P. M., 106; 3 P. M., 112, as she became unconscious. At 6 P. M. her pulse was 158; at 8 P. M. 163; at 9 P. M. it is recorded as 154.]

PERINEPHRITIC ABSCESS, PROBABLY EMBOLIC IN ORIGIN.—OPENING AND DRAINAGE

HISTORY

THE patient is a married woman aged forty years. Her family history is negative for tuberculosis and carcinoma. Her husband and four children are living and well. Her past history is negative. Menstruation began at sixteen years of age, was of the twenty-eight-day type, regular, never painful, and usually of three days' duration, the patient using three or four napkins a day. Her last menstruation occurred April 9, 1914, and was normal.

The patient became pregnant in December, 1913. She continued in good health until February 15, 1914, when, without apparent cause, so she tells us, she aborted. She did not flow after the abortion. No instruments were used and no internal examination made. She was attended by a midwife. She was up on the third day following the miscarriage, feeling in her usual health, and remained perfectly well until about one month later, when, on the evening of March 20, she had a severe chill, which lasted about fifteen minutes, followed by a high fever. The next morning she felt considerable pain in the left loin over the region of the kidney. The pain was not sharp or cramp-like, but dull and boring in character, and radiated all through the upper abdomen. On the evening of March 20 she noticed a tender area on feeling of the left side of the abdomen at the site of the pain. This tenderness has continued. She had no more chills, but the fever continued high—how high she does not know. About one week later the tender area on her back became swollen and the pain increased. About April 10 she consulted a doctor, who advised her to come to the hospital. Ever since the onset of the illness she has felt pain in the left side of the abdomen and in the back, and has had scalding sensations on urinating. She has never passed blood or pus in the urine, so far as she knows. She has been stubbornly constipated since the miscarriage. The stools have always been normal in color. She has had no intermenstrual flow and has never vomited.

Examination.—There is a mass on the left side of the abdomen, extending from the lower margin of the ribs to a point one and a half inches above the posterior superior iliac spine, and from a little on the left side of the vertebral column to the anterior axillary line. The mass is hard, pits on pressure, and is not movable; the tissues over it appear inflamed. Palpation causes severe pain. Vaginal examination is entirely negative.

Laboratory Findings (April 16, 1914).—Leukocytes, 13,600 per c.mm.; hemoglobin, between 70 and 80 per cent. The urine (freshly voided specimen) contains no albumin, casts, or sugar; odor normal; specific gravity 1015; no red cells; the white cells average five or six to the field of a 1/6 dry objective. The stools give a weakly positive test for blood with the guaiac method.

COMMENTS AND OPERATION

Dr. Murphy (April 17, 1914): What is the matter with this patient?

Intern: I shouldn't be surprised if she has a psoas abscess. I think a tuberculin test might be made to clear up the diagnosis.

Dr. Murphy: The trouble began with a chill and an elevation of temperature. There was some soreness in the left loin within twenty-four hours of the onset. Is that the history of a psoas abscess? Does one have to make a tuberculin test to differentiate the fact that the condition is not a tuberculosis? Not at all. The doctor lost sight entirely of the relationship between the urinary symptoms and the pain in the left side. Just a few days after the onset of the pain the patient noticed burning on urination, which lasted for a few days, with no pain referred to the region of the bladder. We have had two similar cases within the last two weeks. Both patients came into the hospital with the history of urinary irritation—burning on urination. Then, after several days, came the severe pain in the loin. Both cases were infections in the pelvis of the kidney. One patient came in diagnosed appendicitis, expecting to have an appendectomy performed. The other patient came in with a rather indefinite history, but her doctor believed the condition to be a gall-bladder lesion. It was only the patient's story in both of these cases that kept me from performing an immediate operation. One patient has already gone home, and the other is ready to go home either today or tomorrow, without any operation in either case, only medical treatment. If we had removed the appendix of the first patient, she would have had no relief of her trouble, but, very likely, an exacerbation of it. If we had drained the gall-bladder of the second patient she would, sooner or later, have had recurring chills and fever, *but without jaundice*, and this combination of symptoms would also have told us to look for a lesion outside the biliary tract. It is better, however, to study the patient's history *before* operation and *avoid* mistakes. Then you'll not have to cover them up later.

This patient's story is not a perfectly clear one. She noticed the urinary irritation only *after* the pain in the kidney set in, not in the beginning of the trouble. Both of the other patients gave us the story of a vaginal discharge, then trouble with urination, and, finally, the pain in the loin. This history gives us just the same symptoms, but in reverse order, if we have received it correctly from the interpreter. The patient herself talks only Italian, and we, therefore, labor at a disadvantage, since we can get her statements only through the uncertain medium of a lay mind, not too intelligent and *sans* education—medical or scientific.

We cannot consider tuberculosis of the kidney in this case because tuberculosis of the kidney does not begin with a chill and does not produce obvious swelling and pain within the first twenty-four hours of its onset. Tuberculosis of the kidney often produces signs of bladder irritation for weeks, months, and sometimes years before local signs or symptoms develop in the loin. Here the patient had local manifestations of renal disease within the first twenty-four hours of her illness.

This condition cannot be a hydronephrosis, because an uncomplicated hydronephrosis does not set in with a temperature of 105° F. and a leukocytosis of 13,600, although there may be, and usually are, pain and swelling, and the urinary findings are not unlike those present here.

A pyonephrosis, too, is extremely unlikely here, because a pyonephrosis would be accompanied by pus in the urine unless the ureter were completely blocked. If the ureter were obstructed she would have pain and retention of urine, the pain being due to the tension on pelvis and ureter produced by the retained urine. The patient did, in fact, have a marked retention of urine. I was called on the phone, at the office, yesterday afternoon, and told that this woman had not urinated for twenty-four hours. Why? Was the ureter blocked or kinked? Probably not, because on catheterizing the patient we removed 300 c.c. of somewhat cloudy urine! There was no odor to it, but microscopic examination showed the white cells present in the sediment to average slightly over 15 to the field of a 1/6 dry objective. There were no casts or red cells found. If this patient had a pyonephrosis with a lump in her loin as large as this one is, she should have not merely 15 white cells to a field, but, more nearly, 1500.

Much more likely than any of the conditions which we have yet considered is a perinephritic abscess. The sudden onset with pain, tenderness, and swelling in the loin, accompanied by high fever preceded by a chill, leukocytosis, and, finally, the development of an exquisitely tender, boggy mass in the renal region, all speak with positiveness in favor of this diagnosis.

Having concluded that we have here a perirenal abscess, we must next ask ourselves its etiology. To determine this point, as a rule, we have to rely largely on the history; but the patient's anamnesis, in this case, leaves us still searching in a fog for the ultimate cause. If we consider first the possibility of sepsis following the abortion to be the cause, we must presume that the infection spread upward by extension retroperitoneally, perhaps along the periureteral lymphatics. An ascending infection may follow this route, whether it originates in the bladder as an acute cystitis and ureteritis, or in the uterus as an endometritis, followed by deeper lying uterine infection, which spreads readily to the retroperitoneal cellular tissue on the side of the uterus where the ureter lies beside the uterine blood-vessels. By a very similar retroperitoneal route a metastatic abscess may form around the left kidney after an acute inflammation in a retroperitoneal appendix vermiformis.

Let us analyze the patient's story again. She had an abortion, cause unknown, about the middle of February of this year. A month following that abortion her present illness began suddenly with a chill. We are unable to elicit any history of palpitation or other cardiac disturbance with, preceding, or following the chill. Then she had a sudden and rather severe pain in her left loin, persistent, aching, and boring in character, not sharp or cramp-like. She had an elevation of temperature to 105° F. The suddenness of the onset, the month's freedom from all symptoms of pelvic trouble after the abortion, make me think that, in all probability, she had a septic infarct in the cortex of the left kidney, possibly originating as a thrombus. This septic infarct, which produced sudden, intense local pain, a single chill, then fever, formed an abscess which evidently did not rupture into the pelvis of the kidney, but into the perirenal tissues. If it had, on the other hand, ruptured into the renal pelvis, the patient would have had large quantities of pus and, probably, blood escaping into the urine. One of the features of the urine when the renal cortex is involved in a septic process, or even when there is only a perirenal abscess, is an increased number of leukocytes in the urine, and occasionally a blood-cell. Large quantities of pus in the urine in these cases are sometimes found, but need not be present, depending on the degree of cortical involvement. If an abscess is in the renal cortex, the most common place for it to rupture, following the line of least resistance, is outside the kidney. That this patient has now a perirenal abscess is certain. That it is of embolic origin is very likely, in spite of the entire absence of cardiac manifestations. It is not necessarily true that the embolus came from the abortion, either directly or indirectly. It could have originated from an infection in any other portion of the body, and yet lodge in exactly the same place that it did. Where the primary focus was, we are unable to say. The patient also gives no history of a preceding bladder infection to account for the extension upward of a ureteral or periureteral infection. She has no distinct history of an infection in the pelvis to account for the upward extension nor the local findings which accompany a pelvic infection. She has merely the history of sudden loin pain, accompanied by acute septic manifestations, a syndrome characteristic of a septic embolism in a rich lymphatic zone, such as we have in and around the kidney.

Some of you doctors look surprised that I speak of the kidney region as one rich in lymphatics. In the days that many of you and I studied anatomy we were taught that the renal region was poor in lymphatics. In recent years, however, Stahr, who has made a prolonged study of the subject, has shown that the kidney itself possesses a very rich network of lymph capillaries, while a second network lies in the deeper layers of the fibrous capsule, and a third and very extensive network lies superficially in the fatty capsule. These two perirenal networks communicate freely with each other, and also with the lymph-vessels of the cortex of the kidney.

Judging by the clinical aspect of the case, the abscess does not communicate with the renal pelvis. If such proves to be the case, we shall simply open and drain the abscess from behind. If the renal necrosis extends high enough to involve the hilum of the kidney, urine will have escaped into the abscess cavity, and urine, as well as pus, will drain subsequently from the wound. It is our opinion, however, that the abscess does not involve the pelvis of the kidney, but only the cortex. Why? Largely because the patient has had only 15 pus-corpuscles to the microscope field, and no red cells. The presence of a slight amount of blood in the stool suggests that the abscess lies close to the bowel, and might perforate into the latter if surgical intervention were long delayed.

The time element in the development of disease is one of the important factors to be taken into consideration in all diagnostic work. The patient's own story is probably the most important single factor in diagnosis, and in the patient's story the time element is one of the extremely vital points, particularly the time relation of the individual symptoms and their order of occurrence. At the recent meeting of the American Surgical Association in New York, and for the first time in all my connection with medical societies, the keynote in the diagnosis of practically all the conditions under discussion at the meeting proved to be the clinical history. In this clinic we have been insisting for a quarter of a century that the patient's own story is of paramount importance. In duodenal ulcer, gastric ulcer, in hepatic lesions, in gall-bladder lesions, the members of the Surgical Association brought out the fact that the clinical history in diagnostic importance takes precedence over all other evidences of disease. The history of this patient of today is extremely important, even though not perfectly typical, apparently, of the condition present.

[Dr. Murphy made the usual oblique lumbar incision through skin and fascia. In cutting through the latissimus and serratus magnus muscles down to the lumbocostal fascia it was noted that the soft tissues were already infiltrated by the inflammatory edema. Pus welled out of the wound immediately, showing that the lumbocostal fascia was incised.—Ed.]

Dr. Murphy: We shall make a vaccine from this pus. There is no odor to it, which means that the organisms causing it are probably not anaërobes. The pocket extends clear down to the kidney, meaning that the suppuration was primary in the kidney and has ruptured posteriorly, producing this burrowing abscess. The abscess, therefore, was probably embolic in origin.

That there is no odor to the discharge would speak in favor of cortical renal origin, whereas if the condition had originated from the renal pelvis or from the intestine, stomach, or bone, there would probably be a mixed infection present and an odor. The rib is not involved, although bathed in the pus.

To summarize: All the soft tissues in the loin were found infiltrated; the pus burrowed under and around the twelfth rib, but the costal periosteum is still intact; the pus extends down to the surface of the kidney, thus verifying the clinical diagnosis of perirenal abscess. We are inclined to believe, from the clinical history, that this suppuration originated from a septic infarct from some unknown source. A large rubber tube was inserted as a drain. As we stated before beginning the operation, the abscess does not communicate with the pelvis of the kidney, for if such had been the case, the urine examination would have shown a great many more pus-corpuscles than 15 to the field of a 1/6 dry objective.

[NOTE.—The wound drained freely and the temperature returned promptly to normal. The patient left the hospital at the end of two weeks, and dressings were continued by her home physician. She has not returned to the clinic for reëxamination up to August 1, 1914.—ED.]

TRAUMATIC EPILEPSY.—ADHESIONS BETWEEN SCALP AND DURA THROUGH AN OLD TREPHINE OPENING.—ADHESIONS BETWEEN DURA AND CEREBRAL CORTEX.—OSTEOPLASTIC FLAP OPERATION AND FREEING OF ADHESIONS.—SUBSEQUENT COMA AND DEATH

HISTORY

THE patient, a man twenty-four years of age, entered the hospital June 15, 1914, with the following history: When four and a half years old (in 1894) the patient was kicked by a horse on the left side of the head just above the parietal eminence. The skull was fractured. A surgeon removed from the skull a broken piece of bone about one and a half by three-quarters of an inch wide. The patient made a rapid recovery from the operation and remained apparently well until three years ago (1911). One night in May of that year he called his father, who came from an adjoining room and saw the son, within the course of two or three minutes, seized with a twitching of the muscles of his face, legs, and arms. The eyes rolled upward. The patient remained unconscious during the attack and for one to two minutes after it had passed.

Since this initial attack the convulsions have recurred repeatedly. His arms and legs sometimes become rigid following the attacks, usually on the right side; he is usually hazy mentally for about twenty minutes after the convulsion, and then drops off to sleep for from fifteen minutes to half an hour, awakening, as a rule, with a headache which lasts for from four to eight hours. He feels very tired after an attack.

The first attack occurred May 20, 1911; the next one on July 4, 1911. The attacks continued, from one to two weeks apart, all that summer and until October, 1911. On October 28, 1911, a cranial operation was performed The cicatricial tissue at the site of the former skull fracture was removed and the skin-flap sutured back into place, so the father relates. For six months following this operation benefit was apparent; but after that time the attacks gradually became less frequent, ranging from three to six weeks apart. From February, 1913, to November 1, 1913, the patient enjoyed complete freedom from convulsions, no particular reason for that freedom being assigned; but since that time the attacks have averaged about one every week or two. From the middle of February to the beginning of April, 1914, the attacks again ceased. Since April they have averaged about two each week. The last convulsion occurred on June 7, 1914.

In June, 1909, the patient was struck in the left temporal region by a baseball. He felt weak for the remainder of the day. He has not been able to stand the direct heat of the summer sun ever since this second accident occurred, because of the headache which the sun's rays seem to cause him.

COMMENTS AND OPERATION

DR. MURPHY (June 17, 1914): What was the date of the original accident?

INTERN: 1894.

DR. MURPHY: Twenty years ago, and his epilepsy did not manifest itself until three years ago! Seventeen years is an extraordinarily long incubation period for a traumatic epilepsy, isn't it, doctor?

INTERN: I do not know.

DR. MURPHY: The most valuable portion of my medical experience has been derived, I believe, from the fact that during all these years of my practice, and in spite of the not inconsiderable labor which it involves, I have not only seen, but also examined carefully, practically every patient on whom I have expected to operate. That is my system. Its significance is beautifully illustrated in this case. This intern is probably as good as the average intern. I might concede that he is a little better than the average if I were not afraid of inflating him with praise. And yet he left out of that history one of the most vital facts in the whole case. He omitted entirely to tell us of the fact that this boy, after being struck with a baseball five years ago, remained unconscious for from twenty to forty minutes after the accident. What is one to say of such an omission?

The patient was struck not at the place where the bone had been removed at the operation fifteen years before, but a little lower and farther in front. That lapse of consciousness, although of only short duration, was a serious sign. Not the fact that the patient was struck with a baseball, but the fact that that blow was severe enough to render him unconscious, means that this second injury was fraught with dangerous possibilities. I have often said that I had rather die than receive a blow sufficiently severe to render me unconscious for a considerable period of time. The late sequences of this type of injury I have observed for many years, and I wish none of them mine. A blow which merely fractures the skull without producing unconsciousness, such as that from a sharp instrument, is not nearly so serious a condition from the point of view of future possibilities as that following the blow of a blunt instrument which renders the patient unconscious.

When this patient was kicked in the head by the horse twenty years ago, notwithstanding the fracture of the skull which necessitated decompression, he was not rendered unconscious; but when he was struck by the baseball five years ago he remained unconscious half an hour or more. He was not first stunned, then conscious, subsequently lapsing again into unconsciousness —the sequence which is so typical of bleeding from the middle meningeal artery. He was rendered unconscious immediately, and when he came out of coma he remained conscious continually thereafter.

This is a typical case of traumatic epilepsy, originally of the Jacksonian type, but now closely bordering on the *grand mal* type. Let me recall to your minds the different varieties of epileptic attacks. The Jacksonian attack begins in one extremity or, less frequently, in two, with twitching, which slowly spreads until it may involve the whole body, and be accompanied by complete unconsciousness; or it may be confined to the original regional twitching, without loss of consciousness occurring.

In the *petit mal* type of epilepsy the patient, perhaps while sitting or talking or being otherwise actively occupied, suddenly but only momentarily loses consciousness. The expression of the face may not change for a minute. After a minute, a half a minute, or even a few seconds, the patient starts doing whatever he was previously engaged in, just as though nothing had happened, because he himself is unaware of the momentary hiatus; and even his immediate family may be a long time in comprehending the significance of these lapses. There may be one or twenty or fifty of these "psychic equivalents" in a day. The patient may be singing and, after a moment's pause, starts again on the same note. He may be playing the piano and, after the interval, strikes the next key correctly. That is the *petit mal* type of epilepsy, a momentary flash of complete coma, unaccompanied by convulsions. The *petit mal* attacks usually pass over sooner or later into the convulsive form of epilepsy. Unlike the Jacksonian, they have no *aura*, or premonition as to what is about to happen, and know nothing of their subsequent mental lapse; but they may have a little psychic haziness or incoherence following the attack. The nature of these attacks is often by no means obvious, either to patient or practitioner, and so they have gained the name of "masked epilepsy."

The *grand mal* attack consists of severe convulsions, which come on usually without a moment's warning, or may be preceded by an aura of some sort, psychic or somatic. The epigastric aura is rather the more common. The patients fall to the ground, wherever they are; there is first a short period of generalized tonic muscle spasm followed by twitchings, which are at once, or soon become, general over the whole body, continue for a certain time, and are accompanied and followed by complete loss of consciousness for a longer or shorter period. After this profound sleep they finally wake up with a headache and some mental confusion or torpor.

We are unable to tell with certainty from this patient's his-

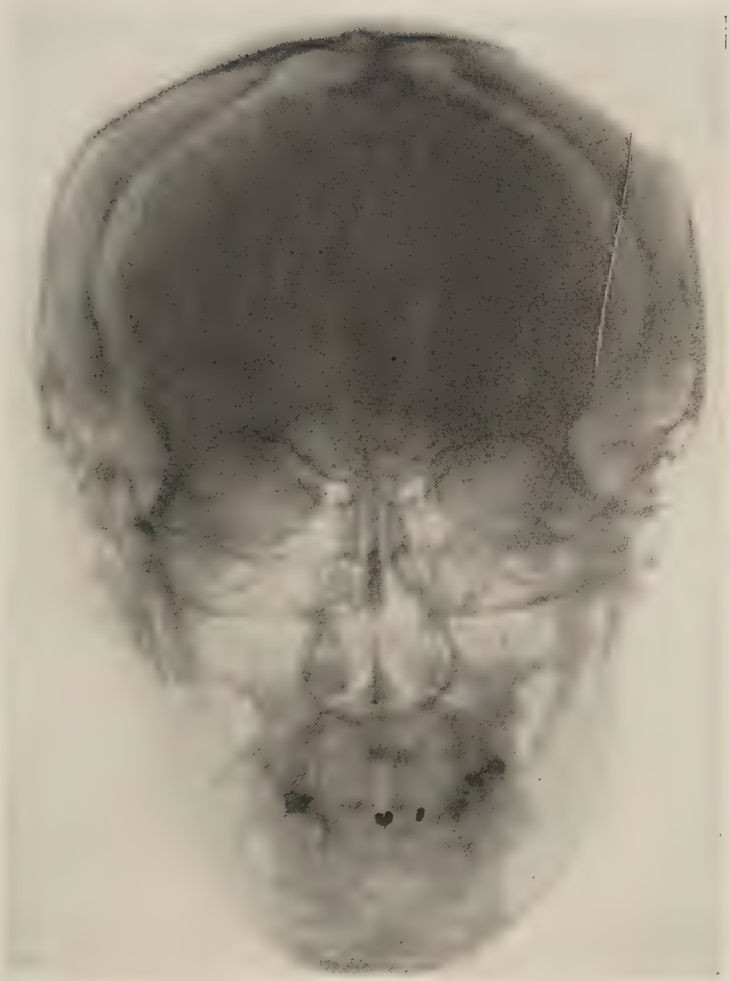


Fig. 270.—Traumatic epilepsy. Adhesions between scalp and dura through an old trephine opening. Adhesions between dura and cerebral cortex. This postero-anterior radiogram shows the cranial defect in the left parietal region, where a piece of bone was removed in 1894 from the patient's skull following a compound fracture of the skull the result of a horse-kick. The adhesions between the dura and the scalp, on the one side, and between the dura and the cerebral cortex, on the other, were situated beneath this opening. In operating, Dr. Murphy's osteoplastic flap included a large area of bone containing this cranial defect in its center.

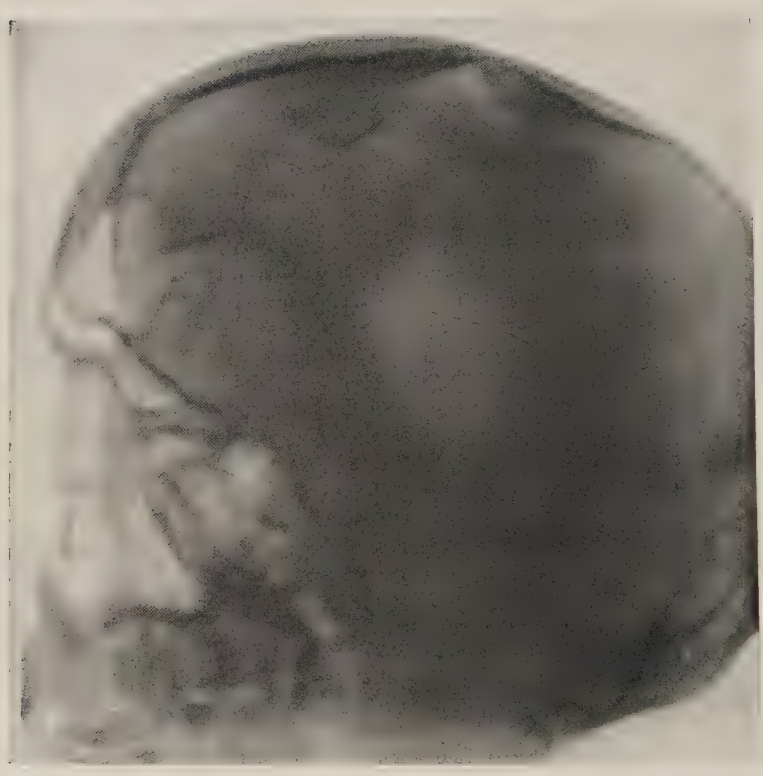


Fig. 271.—Traumatic epilepsy. Lateral view of the skull (right side of face on the plate), showing the cranial defect in the left parietal region. The pointed light area seen just below the vertex of the skull is an erosion made by a large Pacchionian granulation, a not infrequent normal finding.

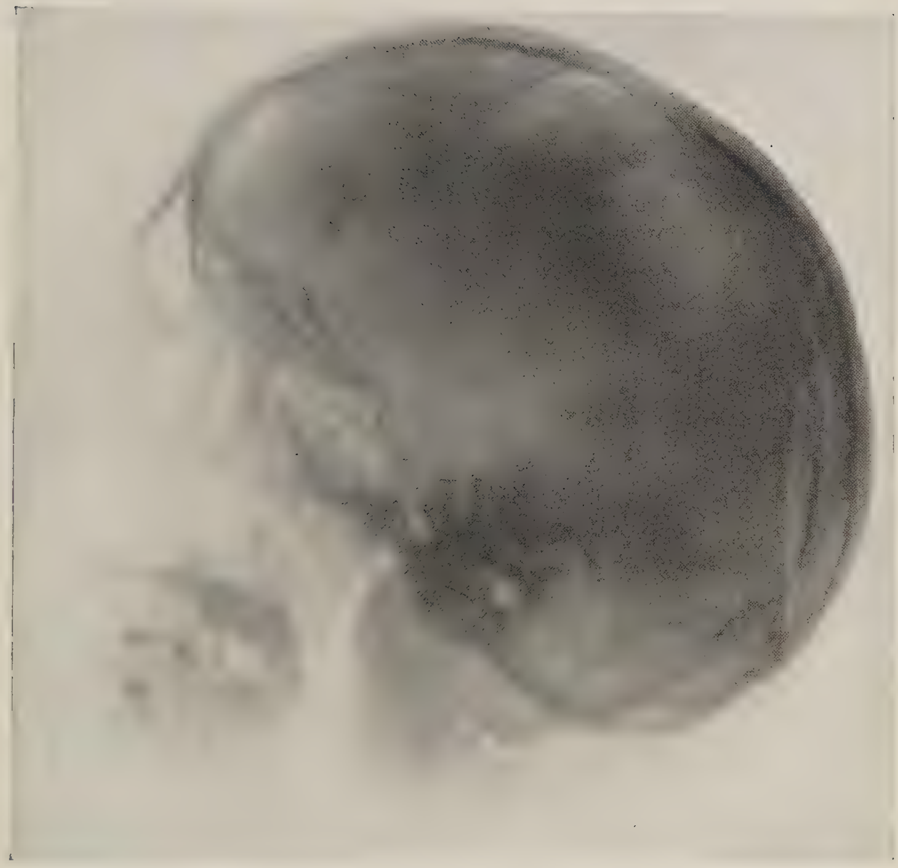


Fig. 272.—Traumatic epilepsy. Lateral view of the skull (left side of face on the plate), showing the cranial defect in the left parietal region.

tory whether it was the baseball or the horse's kick which is responsible for his present condition; but of the two I should consider the baseball injury the more serious. [Outlines the scalp incision by passing Makka's long flexible hemostatic clamps just outside the proposed line of the flap.] We shall make our incision well within these lines of clamps. The bleeding from the scalp is much lessened by passing this long Makka's clamp along the skull in the temporal region, and clamping the overlying scalp with it. The construction of the instrument is simply that of a long flexible spring clamp. [Makes incision down to the skull.] Most of the bleeding is coming from the inner side of the scalp incision, because the patient has on the other side a scar which is relatively avascular. I do not like to operate these cranial cases that have been operated previously. They usually have dural adhesions; and the dura is often so fixed that one cannot get around it well, and is often torn by whatever cutting instrument one uses to open the skull. [Makes the first opening in the skull with the Neff trephine.] I dislodge the dura from the inner surface of the inner table with the dural separator (Braatz sound). Then I start with the shoe of Neff's drill outside of the dura and close to the skull, and endeavor to keep the shoe of the instrument outside of the dura all the time while I am cutting the bone-flap with the drill. When the drill works at all, it works rapidly and effectively. Perhaps it tends to produce a little more diploëtic hemorrhage than the De Vilbiss forceps. The old scar runs clear to the midline of the scalp, to a point over the longitudinal sinus.

[Cuts out a large bone-flap with base downward.]

With the Doyen chisel I must blaze the way in which I wish the bone-flap to break at its base. I wish to keep and return the osteoplastic flap, and, therefore, hold it fast to the galea with the Krause claw forceps. I am likely to tear the dura in elevating this bone-flap, but that does not matter greatly, as I expect to remove a large portion of the dura anyhow.

[Dr. Murphy, in elevating the bone-flap, separated the dura from the under surface of the inner table. In about the center of this bone-flap was the trephine opening made in 1911 by the patient's home surgeon. The bone had failed to regenerate over this defect after the operation, and the dura eventually became adherent to the scalp through the opening. It was, therefore, necessary to separate the organic adhesions between the scalp and the dura in order to raise the bone-flap completely.

There was some venous oozing from the diploë when the bone-flap was broken off at its base and completely raised; but this hemorrhage was promptly stopped by the use of Horsley's bone wax. There was no cerebral pulsation apparent beneath the dura. The dura was split and the brain exposed. Fairly firm organic adhesions were seen between the cerebral cortex and dura. The dura was excised over the extent of these adhesions and egg-membrane was used to cover the raw surface thus produced. None of the cortex of the brain was removed. After the dura was opened the brain showed some pulsation and bulged under considerable tension, apparently, into the wound. The osteoplastic flap was replaced without suture or other fixation except that the wound in the scalp was securely united with silkworm-gut and horsehair sutures. No drainage. A copious dressing of sterile gauze was applied to the wound.]

What are this patient's chances of permanent recovery? About 5 per cent. I should say. Approximately one case in twenty gets well and stays well. I saw just the other day a man on whom I operated some fifteen or eighteen years ago for traumatic epilepsy. The history of his trouble is as follows: He was riding in a sleigh up in northern Wisconsin when the horse ran away and threw him out of the sleigh against a telegraph pole. He sustained a fracture of his skull, not compound, and was unconscious for three weeks. After regaining consciousness he went along without further symptoms for about three years,—I do not remember the exact details of the case now,—but in any event he went along for a considerable period of time, apparently entirely free from any consequences of his injury, before he had his first convulsion. After the first convulsion he had many others in rapid sequence. He came to Chicago and we operated on him. That was in the days before we excised the dura in such cases. We made a large osteoplastic flap in exposing his brain, opened the dura, and found no pathology of apparent significance. What we did for him was practically nothing but a temporary decompression. The wound healed by complete primary union. He went home and for three or four weeks had no convulsions. Then he had an almost continuous succession of them. Dr. Lemke, who was taking charge of my practice while I was out of the city, went up to the patient's home and through our decompression opening tapped the lateral ventricle with a grooved director, thus letting off the intraventricular tension. The patient's convulsions immediately ceased and he regained consciousness. It was some little time before he had another convulsion, and then he came back to the hospital and we operated on him again. We took out at that time a large portion of the dura covering his motor area on one side, a piece possibly once and a half as large as the piece I removed today. That happened a little over fifteen years ago. I remember that it was in 1898, just before Dr. Lemke went to the Spanish war. Since the second operation—the removal of dura—the patient has remained entirely free from convulsions or other symptoms of epilepsy. He went into the real estate business and developed into quite an important man in this city. Within the last few weeks the patient has had two or three slight convulsive attacks—the first in over fifteen years. We sent him to the country the other day, just for a rest. He has been doing very heavy work and bearing much responsibility in connection with his business, and we thought possibly that complete rest might restore to him his usual immunity to these epileptic manifestations.

The period of time which may elapse between the trauma and the first convulsion is very great. One patient I had was a young man who suffered a comminuted fracture of the skull when he was a baby. He fell from a second-story window on to the stone pavement. It was twenty-two years after that injury that the patient had his first convulsion. He was then holding a clerical position in a bank, where he was mentally greatly taxed and had much responsibility. He had developed rapidly, and, in fact, almost precociously, in school in the interim, as many of the epileptics do. He always did work above his grade in school.

In operating on him we took a large area of the dura; but he derived little or no benefit from the operation. Why? Probably because his injury was a comminuted fracture which involved the base of the skull as well as the vertex. He had no depression whatever on the surface of the skull. There was only the scalp scar left to show he had had an injury there. That has been the usual outcome, according to my experience, in the cases of traumatic epilepsy resulting from severe injuries to the skull. Therefore, when the patient has suffered an extensive cranial injury, as from a fall, or when the skull fracture has involved the base, I do not operate for the epilepsy, because operation rarely or never benefits them.

It is the irritation of the dura, I believe, which plays the most important rôle in the production of traumatic epilepsy. It is the removal of the dura over that zone of injury which gives the patients the greatest benefit. The time was when we excised not only the dura, but also the outer layer of the motor cortex, when the latter lay just underneath the pressure area. This more extensive operation did not give so good results as the simple removal of the dura. For a time we used a silver plate to plug the trephine opening. Now we use the osteoplastic flap practically all the time, turning it back into place as we did today, as soon as we are through with our intracranial work. The circulation through the diploë of the bone-flap was very good in this case, notwithstanding that there was a defect in the center of our flap where the bone had been removed at the previous operation and had failed to regenerate. We freshened the edges of this old opening and, I believe, it may fill in with bone and heal over nicely. That area of dura which we removed was irritated by its fixation to the scalp through the old trephine opening in the skull. The egg-membrane which we planted over this area will prevent the development of scar tissue between the scalp and the cerebral cortex, and thus allow new bone to bridge the old operative gap. We hope that the presence of bone will have a less irritating influence on his motor cortex than did the scar tissue from the scalp. Brown-Séquard believed that it was the irritation of the dura which caused epilepsy in these traumatic cases, and he made some experimental demonstrations at Rush Medical College when I was a student, showing that irritation of the dura, in pigeons at least, could produce convulsions. He was then traveling around the world making these demonstrations, and striving to gain a general acceptance of his views. His work impressed me at that time. As I became older and had greater experience with these cases I came to believe that his estimate of the situation was fairly correct. The reason that the basal fractures give such poor post-operative results is because the surgeon cannot control the dura over the base of the brain, because it is inaccessible with our present operative technic.

Let the record show that there were organic adhesions between the dura and the scalp, which we freed, without opening the dura, in the process of turning back the osteoplastic flap; that there were also organic adhesions between the dura and the pia and cerebral cortex; that there was considerable bleeding on freeing these last adhesions; that this hemorrhage was controlled by catgut sutures on fine needles; that we removed an area of the dura measuring about one and three-quarters by two inches; that we covered all the denuded area of cortex with egg-membrane, overlapping the dura with it over its entire circumference; that the osteoplastic flap was then replaced in the regular way; that the boy was ordered put to bed at once with the head of the bed elevated a distance of eight inches; that his head is to be kept elevated in transit to his room from the operating-amphitheater.

[Note.—Thirty-six hours after the operation the patient developed a series of convulsions, went into coma, with a marked rise of temperature. The wound was immediately opened, but nothing pathologic found—no deep hemorrhage nor any sign of infection. The patient died eighteen hours later, never regaining consciousness, his temperature remaining high. No autopsy was allowed.—Ed.]

EPITHELIOMA OF GLANS PENIS.—AMPUTATION

HISTORY

The patient is a married man, aged fifty-five years. His family history is negative for tuberculosis and carcinoma. His past history is negative, except for the measles, which the patient had one year ago. Venereal infection is denied and his habits are good. His wife is living and well.

About three months ago, in January, 1914, he noticed a continual irritation of the penis, which continued for about a week. On examining it he found under the foreskin several small warts—one about half the size of a pea, while the others were just visible. He cannot say whether the growths were attached to the foreskin or the glans penis. He noticed that the largest of the warts had disappeared in March, 1914, and at its site there was a small ulcer. During the same month he noticed that the other warts were beginning to ulcerate, and that if the urine touched them they burned. During the first week in April the foreskin became tight and swollen. He consulted a doctor, who said a circumcision was necessary. On Tuesday, April 14, 1914, the doctor split the foreskin on the dorsal aspect of the penis and discovered under the foreskin that the whole of the glans penis was eroded by deep ulcers which bled easily. He amputated the foreskin. A section of this pathologic tissue under the microscope showed it to be an epithelioma. The patient says that he has had no pain whatever and only a little discomfort from the tightness of the foreskin, while he has had itching and burning whenever urine touched the affected part, but there has been no bleeding. He has had no difficulty in passing urine, and has lost no weight. His appetite is good and his general health apparently good.

Examination on admission to the hospital shows the glans penis and foreskin eroded. The involvement appears to be secondary on the foreskin. The inguinal glands on both sides are palpable. Microscopic examination of an excised piece of the tumor shows the pathologic condition to be an epithelioma.

COMMENTS AND OPERATION

Dr. Murphy (April 18, 1914): From the slide that is being passed one can see that the growth is an epithelioma. It is sad that the man allowed his condition to go neglected so long before calling a doctor's attention to it. When the foreskin was turned back, practically all the glans penis was found to be destroyed, and the under surface of the foreskin was also eroded. The epithelioma began, we believe, on the glans. The growth was excised, and the surgeon brought us a very good slide showing it to be an epithelioma. The patient must now have a high amputation of the penis; then he must have all the glands in the inguinal regions on both sides removed—an operative procedure which is very extensive, but necessary to prolong his life. If this were an adenocarcinoma instead of an epithelioma, we would say that the hope of saving his life would be practically *nil* at this stage of the disease. Because of the involvement of the inguinal glands it is possible that the malignant epithelial cells have already passed through the glands and entered the circulation and may now be growing in some other part of the body. A carcinoma arises on the foreskin usually from the mucosa, which has a relatively sparse blood-supply. Therefore, it rarely ever has a vascular transmission. When the growth begins on the glans, however, because of the very rich vascularization here, it not infrequently leads to vascular transmission to other parts of the body, one of the dangers of malignant disease beginning as an ulcer on the glans or as an ulcer in the urethra. The same general rule holds true with the chancre beginning in the urethra or well forward on the glans penis. Practically always when the chancre begins in the urethra the spirochetes go directly into the blood, so that local measures are of no avail to abort the disease. Why I say that is because, although we have known about the spirochetes only a little time, we have known about the clinical course of chancre of the urethra for a long time—in fact, about as long as we have known much about venereal disease. A patient who has a urethral discharge which runs four, five, six, or seven months, and has also a nodule in the urethra, as a rule does not have the secondary local manifestations of chancre—the adenopathies. The spirochete goes directly into the blood, produces mild general symptoms which are easily overlooked by the patient, and it is only years later, when the infection appears as a spinal cord—or a cerebral—lesion that one asks the question, "Did you ever have syphilis?" The patient will honestly and firmly reply, "No." But if you go into his history you will find that he had a "gonorrhea" of long duration, and he may be able to tell of the nodule. Then you make a luetin or a Wassermann test, and get a positive reaction. That makes you feel pretty certain that that patient had his chancre in the urethra, the more so if he gives no history of a mouth or throat trouble resembling a chancre.

You can see here the place on the penis where the tissue was removed for microscopic examination. We must perform an amputation of the penis high up, and then excise all the infected glands. The danger here is of getting a cellular infection in the site of the inguinal dissection from the recent wound on the penis; and if we do, it is likely to give much trouble. We are not putting iodin on the scrotum, because it not infrequently produces blisters there which are very painful.

We shall put the patient in the Trendelenburg position because it gives a much better view to the visitors in the seats. [Incision.] There is the dorsal chain of lymphatics through which systemic infection most often occurs. The next step is to free the urethra for a considerable distance laterally, as we wish to preserve the corpora cavernosa. I am dissecting away with the urethra the corpus spongiosum, keeping close to the wall of the corpus cavernosum as I free it. Now we have the urethra, standing out with the corpus spongiosum entirely free from the corpora cavernosa. Now we are dividing the corpora cavernosa. We close up the cut end of the corpora cavernosa accurately and then fix the urethra to the stump. Sutures fasten the wall of the stump of the urethra and corpus spongiosum to the stump of the corpora cavernosa, so that the urethra cannot retract into the stump. Now I split the end of the urethra, which I have purposely left long, and attach it to the skin, so that there can be no retraction of it, and so that the stump of the penis will be covered entirely, either by urethral mucosa or by skin.

Carcinomata of the penis have a great mortality, one reason for which is that they are usually treated on the expectant plan. That means expecting them to "get ripe" before picking them off, in place of treating them early on the radical excision plan, which should be the method for all these malignant neoplasms of the penis, just as it is for those of the breast, uterus, lip, tongue, and other organs. If we could only perform these operations in time, the patients would have a very much lower cancer mortality. It does not seem to me that we are making very much progress—that we are always waiting and procrastinating. It is the custom with a growth of this kind, when it is as large as a pea, to wait until it becomes as large as a nickel, "to see what it will become"; but it is the same malignant disease all the time, whether it be small or large. We have often had occasion to refer to a lesion of the breast which resembles this lesion very much, from the fact that it does not destroy tissue rapidly, but ultimately destroys the patient, and that is Paget's disease of the breast, which should be called Paget's cancer. Paget's disease first shows as a little crust on the nipple and then as a larger crust. Then the lesion ulcerates and attracts notice, all the time increasing in size and spreading. It finally goes to the surgeon for excision, but usually too late to save the life of the patient. The growth was a carcinoma when it was of wheat-grain size, just as much as it is when it is of dollar size. The important difference is that the larger lesions have been given time to produce metastases. The patient and the doctor may be idle, but the cancer is always working.

Let the record show that the glans was entirely destroyed, the destruction having extended down into the corpus cavernosum.

I am now fastening the corpus cavernosum to the skin, so that the skin will not be drawn too far in by the retraction of the urethra. Now I am covering over the remaining raw surface by swinging over a skin-flap. We hope to get coaptation and primary healing of the wound clear up to the abdomen. I am excising the corpus cavernosum a little farther so as to make an indentation of the skin ample to support the urethra.

We shall not remove the inguinal glands at this time, because dissecting out the glands in a patient with this large quantity of adipose tissue would mean an enormous opening up of cellular spaces, which would stand in great danger of infection, in view of our recent handling of this ulcerated growth which we have just excised. If we get a primary union of this wound, it will be only a few days until we can take out the glands, being able to take them out then with practical safety against infection. We are fixing the urethra fairly firmly to the surface of the skin, it being desirable for the comfort of the patient subsequently that it should come well up to the surface, thus preventing the development of the dermatitis, which is so prone to occur on a skin surface frequently wet with urine.

The mixed infection which has been present on the ulcerated surface of this tumor is very likely a material factor in the enlargement of these glands. There may be cancer metastases present in the glands in addition to the inflammatory swelling, and then, again, there may not. I am inclined to the latter, and hopeful, view. Now that we have anchored the urethra and complete the closure of the wound, we should insert a permanent catheter in most cases. But this patient is fifty-five years of age and has already had some irritability of the prostate. If we irritate it more with a catheter, we might have some serious trouble from it. We shall, therefore, not use a permanent catheter in this case but shall treat this wound just as we would treat a lacerated perineal wound. After each urination the field will be washed off with lysol, carefully dried, and then covered over again with bismuth subiodid powder.

[Note.—The wound healed by primary union except for a small focus of urinary infiltration close to the urethra. He had no difficulty in urination, and a catheter was not used. The patient left the hospital May 10, 1914, with the wound completely healed. He is to return later for the removal of the inguinal glands on both sides.—Ed.]

CARCINOMA OF THE CORONA PENIS WITH METASTASIS IN THE INGUINAL GLANDS

HISTORY

The patient is a married man, aged forty-nine years. His family history is negative as to carcinoma. He had a chancre at nineteen and gonorrhea at twenty-five years of age. His wife has had no miscarriages and has borne him two healthy children.

Two years ago, in January, 1912, the patient noticed on the penis a small red area at the side of the corona. This spot grew during the ensuing year to the size of a five-cent piece. Then it partially healed for a time. The lesion has become slightly smaller now than it was. It has slightly elevated borders, is triangular in shape, one-half inch long, and one-fourth inch wide. It is constantly itching, but never painful. There are small hard elevations like vesicles over its surface, particularly at its border. Often a clear fluid exudes from the lesion, but it never bleeds.

In December, 1913, while bathing, the patient noticed in the upper right inguinal fold a small tumor, about the size of a marble, which was soft and red. This tumor has grown gradually until now it has become the size of a goose-egg and is hard in places. In the last week of February, 1914, he noticed in the lower inguinal fold another small, red, soft mass, not painful. Now the latter has become hard and tender and is the size of a walnut. He says he has had no chills or fever during this time. He denies recent venereal exposure. The inguinal swellings have never disappeared even temporarily, nor have they ever diminished at all in size since they were first noticed.

COMMENTS AND OPERATION

Dr. Murphy (April 8, 1914): Did the lesion begin at the site of the former chancre? The history should record this point. When was the lesion on the penis first noticed?

Intern: In January, 1912. I do not know where the former chancre was.

Dr. Murphy: You say the lesion on the penis became smaller?

Intern: Yes, sir.

Dr. Murphy: No, doctor, you did not get that right. The lesion did not *become* smaller. I wish to emphasize that point.

Intern: He said it did.

Dr. Murphy: You did not get it right. You should have found out what *made* it smaller. What was done to it? Why do I ask that question? Because if this lesion *became* markedly smaller spontaneously, that is positive evidence that this growth is not malignant. If it healed over spontaneously, you could say positively that it was not a malignant lesion; but if it healed over after the actual cautery was applied, the diagnostic significance is entirely different. You make the differential diagnosis between lupus and carcinoma ordinarily by what? By the fact that in lupus there are some areas of healing while other areas are breaking down. In epithelioma, with the exception, very rarely, of the rodent ulcer or basal-cell epithelioma of the aged, you never find areas of healing. The cancer may scale over, but it does not heal. The course is somewhat different, however, when you put glacial acetic acid on such a tumor. Following the destruction of the tumor tissue by the caustic, you may have repair occurring in the normal way. How many times was the sore cauterized?

Patient's Physician: Twice.

Dr. Murphy: There is the primary lesion on the penis. The moment you look at it its appearance makes you suspicious of malignancy. He has had it for two years, and in the beginning it was larger than now. It was cauterized twice with the actual cautery. When was the last cauterization?

Patient's Physician: About five months ago.

Dr. Murphy: I shall take out all of that neoplasm. What date was it that he noticed the lump in the right inguinal region?

Intern: December, 1913.

[Excision of the small tumor on the penis, together with a small encircling area of normal tissue of the glans and corona.]

Dr. Murphy: I shall send this specimen to the pathologist for examination at once. Our diagnosis of this lesion on the penis is that it is malignant and that it is an epithelioma. What is this lesion in his right groin, doctor?

Intern: A metastatic carcinoma.

Dr. Murphy: If so, then it is necessarily a solid tumor, is it not? But this is not a solid tumor. It has fluid in it. In fact, there is a good deal of fluid. I thought I should get you on that point. I have been in there with a hypodermic needle and I was able to withdraw fluid. What is the matter with that groin now? If that is a metastatic epithelioma, it should make a solid tumor in his groin, should it not? But it is cystic. What is the matter with him?

Intern: An infection.

Dr. Murphy: I thought I could lead you astray there. The fact is that it is not necessary that the growth should be solid in metastatic epithelioma. Where the tumor originates from squamous epithelium, the secondary neoplasm may be cystic, as in epithelioma on the dorsum of the foot, whence you often have the carcinoma metastases cystic in the groin. These secondary neoplasms may break down in the center by liquefactive necrosis and thus become cystic. The fluid in these foci of softening is milky in appearance, but has not so many fat-globules in it as you find in milk. It is my conviction that this neoplasm in the groin is a secondary carcinoma of the inguinal lymph-glands undergoing cystic degeneration. Sometimes when these neoplasms break down in this way they may so block the lymphatics with necrotic material that no further extension of the growth occurs, and removal of the local focus may enable the patient to go years without a recurrence.

[Inverted U-shaped skin incision in the right groin.]

The incision goes clear down to the cribriform fascia at the distal extremity of the wound. We cut down to the aponeurosis of the rectus and external oblique, being careful not to compress the tumor at all. I am cutting wide of the tumor. There is the aponeurosis of the external oblique. I shall clean away everything above the aponeurosis down to Poupart's ligament.

I am blocking the lymphatics, you see, by this method of procedure. There are the vas and the cord vessels standing out. They are not involved, apparently, in the neoplasm, and we must separate them carefully from it. There is the inguinal lymphatic tract passing down over Poupart's ligament to the femoral lymphatic tract to the position where the lymphatics go in at the saphenous opening.

This tumor mass passes beneath Poupart's ligament into the deep chain of glands. Therefore, I must divide the ligament transversely to continue my dissection. Now, I come down to the femoral vessels and I must carefully avoid incising them. Now we are down at the base of the growth. There is no longer any question in my mind as to what the tumor is. It is easy now to tell from its gross appearance that it is a secondary carcinoma. You do not need the pathologist's report now to know that. I am going to leave the center of the wound wide open for *x*-ray treatment. There is no visible tumor left there now. Yesterday's diagnosis is confirmed by today's findings.

The pathologist reports that the frozen section shows an epithelioma of the penis. That is correct. Now the whole picture is clear. This is a very instructive case, because although the intern's errors in the history threw doubt on our diagnosis, yet by following the physical signs closely we were able to come to the correct conclusion—that the growth was malignant. It is an advantage to examine one's cases and get the correct mental picture before you turn them loose on the intern. That part of the story which most suggested the malignancy was that the growth recurred after the first and also after the secondary cauterization. So far as the life of the patient is concerned, this affection will terminate it sooner or later, notwithstanding our *x*-ray treatment and everything else we shall do. The femoral vessels are not involved nor is the deep lymphatic zone. If these structures were involved, we should have had to continue our dissection up to the renal vein in order to be certain of excising all the infected glands.

Let the record show that there was a large cystic and solid carcinoma metastasis situated in and above the cribriform fascia and in the adjacent lymph-glands.

There is the femoral artery. That is a 5 per cent. solution of carbolic acid applied to inhibit the growth of any cells which may have escaped into the field and are lying free. We shall put some nosophen gauze here in the middle of the wound to catch the wound secretion. We shall start the *x*-ray treatment tomorrow.

When the carcinoma commences on the corona penis, you often have this condition present. The corona is where the growth commenced in this man; then it extended over to the prepuce and kept spreading slowly in the proximal direction. Just as soon as the tumor extends on to the prepuce, the patient may get the lymphatic manifestations. So long as it remains confined to the corona lymphatic manifestations are rare, because the abundant lymphatic supply of the penis extends just to the corona, the lymph-vessels of the glans being few and small. Just as soon as the growth reached beyond the corona in this patient he began to show the manifestations of metastasis in his groin.

Carcinoma of the penis, of which epithelioma is by far the most frequent variety, makes up about 3 per cent. of all carcinomata in the male, according to most statistics.

What its ultimate mortality is I cannot state in figures. At least it is very high, largely, I believe, because the patients do not seek the surgeon's aid until the vascular portions of the penis have already been encroached upon by the growth. Of course, as soon as the regional lymph-glands are involved, the prognosis becomes gloomy indeed.

For those who are interested in the subject of the pathology and the spread of carcinoma of the penis I should like to recommend a classic article, that of H. Küttner, entitled "Ueber das Peniscarcinom," etc., in v. Bruns' *Beiträge zur klinischen Chirurgie* for 1900, vol. xxvi, pp. 1–79. This careful study of the subject, based on 60 cases from the author's own clinic, has formed the basis of most of the text-book accounts of the disease written since that time.

Pathologist's Report (*Dr. Zeit*).—The penis removed at operation shows a squamous-cell carcinoma of the glans; and the excised lymph-glands of the groin show extensive metastases of the same cell type.

[Note.—The wound on the penis healed by primary intention. The infected wound in his groin was left partly open for *x*-ray treatment, which is still going on at the present time, May 8, 1914.—Ed.]

FECAL FISTULA

HISTORY

The patient, a young married woman aged twenty-three years, entered the hospital with the following history: There is no tuberculosis in her family. Her mother died of carcinoma of the uterus one year ago (in 1913). She has had no past illness of any significance, excepting an operation for peritonitis in February, 1914. Menstruation began at the age of thirteen, and is usually of four days' duration; the patient uses three or four napkins a day and suffers no pain. Occasionally the periods have been somewhat irregular, coming on two or three days before due, and at other times two or three days after due. She has been married three years and has one child, thirteen months old. She has had one miscarriage, which occurred about January 13, 1914; to it the patient ascribes her present trouble.

About five days after this miscarriage she had some chilly sensations, some fever, and headache. She was taken to the hospital and the uterus cureted. She returned home and went to bed, remaining there for about two weeks, during which time the headache continued, and, she thinks, the fever also. At the end of the two weeks she began to suffer severe pain in the right lower quadrant of the abdomen. She then had high fever, tenderness over the right lower quadrant, and throbbing pain. The doctor who was called told her she had "blood poisoning," and that she would probably have to have an operation. The high temperature lasted about one week, then returned to normal, and after it had been normal for a week she was operated. The date of the operation was February 27, 1914. She thinks the surgeons took out the right ovary, tube, and appendix. A drainage-tube was inserted and pus drained from two separate openings in the line of incision. One opening continued to drain pus until it closed, six weeks after the operation. The other sinus began draining fecal material after the operation, and has continued to do so up to the present time. She has repeatedly noticed berry seeds coming from this sinus, and, therefore, feels certain of the fecal origin of the discharge.

COMMENTS AND OPERATION

Dr. Murphy (June 19, 1914): How was the miscarriage produced?

Intern: She says it was induced.

Dr. Murphy: By what?

Intern: She would not tell me.

Dr. Murphy: It was done with a catheter. Put that point in the history. She did not divulge to me whether she or some one else did it, but that is a matter of relative indifference, so far as our task is concerned. The important fact for us to know is that the deed was done with a catheter; and that seems to be the instrument usually employed in these cases, whether the patient wields it or the midwife or the doctor. How soon after the operation did fecal matter commence to come out of the drainage opening?

Intern: I do not know, doctor; the history does not state.

Dr. Murphy: But it *should* state. Did it appear on the next day or on the second day or the third day, or did it take ten days or two weeks for the fistula to develop? Was it the sequence of the surgical procedure or was it a sequence of an ulcerative process in the bowel? That is the information which one wants in this case and that is what the history should show. We asked her that same question at the office, and she told us that the fecal flow began on the third day after the operation. Then we knew that the surgeon was probably not to blame: that it was the pelvic infection which caused an area of necrosis in the bowel.

Those of you visiting doctors who were kind enough to stay with us the other day during the tedious ordeal of just such another case may not be so ready to accept the same kind of a dose again today. The operation was irksome and very monotonous, but its result has been sufficiently gratifying to more than compensate us for the effort. The patient's condition is splendid. Her pulse is 100 and her temperature 99.4° F. this morning. You will remember the appalling dissection we had to make, the many sinuses present, and the peritoneum wide open to infection. Do you also remember the scheme we followed out? To it our gratifying success and the absence of infection today are due. We shall work according to the same plan again today—first plugging up the sinuses, then cutting around them, excising the cicatricial tissue in the abdominal wall, and thus protecting the field of operation—including the peritoneal cavity—from fecal matter. That is always the plan of campaign which we follow in the operative management of these cases.

The patient's history is a fairly typical one of an infection following an induced abortion and treated by curetment. The average curetment is done almost always with a sharp instrument; and a sharp curet abrades the surface of the uterine mucosa, opening up the lymphatic spaces of the submucosa and muscularis, and simultaneously inoculating them with infectious material. Brisk infection frequently sets in, and with that infection comes first the chill and then the rapid rise of temperature.

When the infection passes out along the lymphatics into the cellular tissue of the broad ligament, it usually forms an abscess there which extends along the tube. This abscess, if not opened by the surgeon, may open spontaneously into the peritoneum, bowel, or bladder. It may burrow rather extensively subperitoneally, and is not infrequently complicated with thrombosis of the adjacent vessels and a pelvic lymphangitis.

[Dr. Murphy stuffed up the fistula's cutaneous opening with a strip of gauze packed in as tightly as possible. A circular skin incision circumscribing the opening, and then a little dissection, freed the end of the fistula, so that a volsellum clamp could be applied to it tightly, closing it and preventing leakage from the sinus during the operation. The cutaneous incision was extended straight upward parallel to the median line of the abdomen, with the aim of making the peritoneal opening eventually paramedian also, and thus protecting the two incisions, after closure of the wound, with the rectus muscle.—Ed.]

I am going through the aponeurosis of the rectus muscle and endeavoring to separate the rectus fibers from the scar tissue and displace them outward from my line of incision. Now we have the muscle-fibers all on the outer side. [Dissects out the fistula further toward the peritoneal cavity, wraps it in gauze, and puts on a clamp to hold it.] That takes care of that infective material. [Dissects carefully downward, hunting for the peritoneum. Finds it, picks it up with tissue forceps, and opens it cautiously on the slant.] I am fortunate in finding that parietal peritoneum so quickly, free from adhesions. [Opens the peritoneum widely, tampons off the free peritoneal space, and begins to isolate the fistula and its attached loops of bowel preparatory to bringing them out on to the abdominal wall.] There are many loops of intestine united here to this fistulous tract. The same scheme which we followed the other day we shall follow now. We must go all around the fistula, with its adherent bowel, encircle the adhesions, and isolate the entire mass. We are gradually working around the point of fixation of the mass to the abdominal wall, so as to get the affected intestine free from the surrounding sound loops of bowel. That is the first and all-important procedure in every type of fecal fistula operation. Here is a suspicious-looking object. It is difficult to say whether it is the granulation tissue of the sinus or the mucosa of an embedded coil of intestine. [Dissects it out.] It is evidently a granulating sinus. I am trying to keep the line of cleavage between sound and diseased bowel here in view all the time.

In getting this mass of scar tissue, sinus, and attached intestines free from their surroundings, it is very important for the operator not to get lost in the extensive dissection which is oftentimes necessary. It is all the easier to lose one's bearings because of the difficulty in recognizing anatomic landmarks here when displaced, as they often are, and when changed in appearance as the result of old inflammatory processes. In this pelvic dissection which we are making our chief difficulty is in getting and keeping our bearings as we proceed. Here, at last, is a structure which appears to be the urinary bladder. It is. It is the first landmark which we have found. It should be easier to find others now. [Dissects further.] Now we have found the uterus and are gradually freeing it of adhesions. Here is the stump of the right tube. The salpingectomy was performed at the previous operation.

Here is the intestine which was involved. We now have it all loosened from its pelvic adhesions. There is not much danger of this patient developing a peritonitis, either pelvic or general, because she has had so much inflammation already in her lower peritoneal cavity that the lymph-spaces must be well coffer-dammed. [Finally frees the fistula with the involved intestine from its intraperitoneal attachments and brings the whole mass out on the surface of the abdomen. It is well isolated from the peritoneal cavity by towels and tampons. Then, loop by loop, the intestine, which is adherent to the fistulous tract, but does not open into it, is freed, and the raw surfaces thus produced are covered by infolding or by approximation to adjacent loops.] Here is a communication between the bowel and the fistula. I am going to get between that and this other fistulous tract before I open either of them. [Isolates both. Then slits up one sinus until he comes to the opening into the bowel.] One dares not put any traction at all on such a connection for fear of making a bad tear. It must be dissected loose—not pulled. Let me see if I can come up behind this loop of bowel and separate it from the other direction. These coils are all matted together on one side as well as on the other. It is not a matter of great choice from which direction the attack be made. Here is one coil which is involved in the wreck of another. Whether there is still another opening or not from the bowel into this sinus I do not yet know. I must first try to isolate it further. Here is another coil which comes into it from the side and is very closely adherent to the sinus. Whether it, too, opens into the common cloaca in the way I mentioned yesterday we shall soon find out. We appear to have a second sinus coming up here to the common suppuration point. We must free that, too. [As he frees the second sinus it is seen to join with the first to form a common cloaca.] See the line of cleavage! There is the common cloaca. Here is a pocket of granulation tissue close to the junction.

[Continues the dissection and frees a third opening into the common cloaca.] Finally, you see, we have arrived at still another one. Let us see if this third opening goes through into the bowel. Yes, it is patent. We have, therefore, three loops of bowel opening into this common cloaca. That practically clears up our problem in this case. Our further procedures now will be purely mechanical. I think I told you yesterday about one such patient of ours who had 25 perforations in the bowel, all leading into a common cloaca.

[Frees one perforated coil from the sinus and closes the bowel opening with two rows of fine catgut sutures.]

I am just a bit shy about the security of that closure, and I shall not take any chances on it. There is no evidence that it would leak; but there is a granulating surface there which it would be well to cover in. In these cases one always uses catgut and not linen for the intestinal sutures, because one expects suppuration; if one uses linen in the presence of pus, an operation for its removal may be necessary before the sinus will close.

I am trying to get this next coil of intestine free from its upper attachment, which does not open into the sinus, and yet not free it from its lower attachment, which does so open. Here are the cloaca and sinus, you see. I seem to be having as much difficulty in distinguishing the proximal and distal ends of these loops of gut as the Irishman did in trying to find the front end of the elephant. It is very essential that I recognize correctly this matter of direction while I am covering over these raw intestinal surfaces, because it would not do to approximate two coils side by side in opposite directions, and so have peristalsis in one oppose peristalsis in the other.

We must next determine where the opening is in this coil and what is its character. Here is where the end of the loop folds on itself. Gradually we have dissected it free from its part of the fistulous tract until we now have it clear out of the field. (This loop had no opening into the fistula.) We are gradually getting along with our task.

We shall next free this sinus of adhesions. That is clear at last, and we have *this* opening in the bowel well exposed. Here is the scar tissue mass about the sinus which is holding the intestine down to the sinus. We shall excise that. Now we must close the opening in the bowel. We are sewing it with a von Eiselsberg needle. That is the best needle for intestinal work. It is made very gradually increasing in diameter from the point to the eye, separating, not tearing, the tissues, and thus avoiding subsequent leakage. That is an overlapping side-to-side suture which we are making. We are infolding the entire transverse peritoneal surface of the coil. That is cicatricial tissue where the needle pulls through the bowel with so much difficulty.

Let the record show that this sinus was closed with two rows of iodin catgut.

Now we come to the next problem—another perforation. This last opening in the bowel makes a large wound. Its greatest diameter is transverse. It is easy now to recognize how impossible it would have been ever to handle such a great defect properly with the bowel down below in the pelvis. It was only by dislodging the entire mass of fistula and attached coils of intestine up here on to the anterior abdominal wall that we became masters of the situation. This opening in the sigmoid is so large that we cannot safely close it, as we did the other two openings, simply with two fine catgut sutures. We must reënforce the closure with a Pagenstecher linen suture. This is the Czerny portion of the suture which we put in first. It includes all the layers of the intestine and is of Pagenstecher linen and will hold firmly. We are placing this first line of sutures in the transverse diameter of the intestine, so that we can infold the bowel as much as possible. We shall not close the lumen of the bowel enough by infolding, however, to produce any obstruction.

Now that the linen suture is in place, we must turn it in and bury it with a second suture of catgut. To do so we start behind this first line of sutures and put in the second row of stitches parallel to the long axis of the bowel, like mattress stitches. In inserting this second row we endeavor not to get mucosa, but to go through the muscularis with our sutures. Next we put in a second row of fine catgut stitches to bury the first; and then we are practically finished. We have still only to snip away the fragments of the sinus from the abdominal wall.

That was a nasty job; but, you see, we succeeded in getting it completed in fairly good shape.

Let the record show that we found three intestinal communications with the cavity of suppuration: two smaller ones coming from coils located low down in the pelvis, and one large opening through which came most of the feces poured out before the operation; that all three openings were closed, the two smaller ones with double rows of catgut sutures, and the larger one with a Czerny stitch of Pagenstecher's linen and two embedding rows of catgut.

Let the record further show that the largest hole was in the upper sigmoid; that there was another hole in the sigmoid lower down and one in the caput coli.

[The abdominal wall was then closed in the usual way, leaving a rubber tube about the diameter of the little finger draining the pelvis through the lower angle of the wound.]

We are putting in these figure-of-8 stitches because we rather expect the wound to suppurate, and wish to prevent any subsequent gaping of the wound and visceral prolapse. The layers of the wall are all so matted together that we are putting in these figures-of-8 as through-and-through stitches, including the peritoneum and the skin, without paying any attention to the anatomic layers of the wall.

Let the record show that Sister Ludwina reports the sponges counted and correct; that on the first count one sponge was missing, but that it was found rolled up in another sponge. Let the record further show that the operator made a careful inspection of the field to determine that no sponges or instruments were left.

We have lost only one sponge since we started to keep a systematic count of them, and that one was never accounted for. We *do* know that it was not left in the patient. We lost one other sponge in an operation on the bladder. We reopened the bladder at once and demonstrated that it was not there. Then we found it later in the hallway just outside the amphitheater. It must have stuck to somebody's shoe and thus have been carried out.

We shall put this patient in the sitting-up position as soon as she leaves the table, so that all the peritoneal secretions will drain into the pelvis. We have inserted a peritoneal rubber tube drain to the bottom of the pelvis to take care of the wound products.

Let the record show that most of our work here was done along the line of peritoneal adhesions and entirely outside of the abdomen, and only a very little work in the true peritoneal cavity after we had first opened the peritoneum to get our bearings.

Let the record show that the large tube-drain was placed in the bottom of Douglas' pouch, extending down behind the uterus; that the patient was ordered placed in the sitting position in bed; that nothing is to be given her by mouth at present; that she is not to have proctoclysis because of the danger of the salt solution working its way through the openings in the colon which we have just closed; that if she shows any material shock after this operation, which she should not, we shall give her an intravenous infusion of normal saline; that if she becomes very thirsty, we shall give her saline by hypodermoclysis.

[June 30, 1914: The wound was resutured.

July 7, 1914: The abdominal wound is all healed.

July 10, 1914: Dressed. Stitches not yet removed.

There was complete primary union after the second suture.]

OLD INVERSION FRACTURE OF THE ANKLE. OPEN REDUCTION.—EXTRA-ARTICULAR NAILING OF THE FRAGMENTS

HISTORY

The patient, a twenty-year-old girl, entered the hospital June 2, 1914, with the following history: About two years ago (July 25, 1912), while out buggy riding, her horse ran away. The patient jumped out of the buggy and fell, striking on her right foot, receiving a compound fracture of the ankle. The foot was turned inward and backward. The leg and foot were put up in a board-splint for two days, and after that a plaster cast was applied and kept on for seven weeks. A sinus developed at the site of the compounding of the fracture and discharged what the doctor told her was serum and not pus. This sinus remained open for about three months and then closed. Last July (1913) it again opened, discharged the same serum, and closed again about last Thanksgiving (1913). She has been using crutches until three weeks ago. There has been much pain in the ankle, especially at night or after walking about on it. The pain has not been so severe during the last two weeks. A skin-graft, which was performed last October, healed rapidly. At present the patient is incapacitated because the foot, being fixed in an inverted position, has developed callosities on its outer side which are painful on walking.

Physical and *x*-ray examination of the foot by Dr. Murphy on June 2, 1914, showed that there is a bony ankylosis between the astragalus and the tibia; that the bony ankylosis cannot be overcome by manipulation; that the best result that can be obtained is to straighten the leg at the ankle and not increase the ankle motion; that the skin over the external malleolus is a mere film; that, therefore, the fracture will be necessarily compounded at the time of the operation, which will somewhat hazard the final result and increase the risk to the patient; that these facts have been fully explained to the patient, and that she has consented to the operation on this basis.

COMMENTS AND OPERATION

Dr. Murphy (June 4, 1914): Look at the x-ray plate—that tells the story. But it tells an entirely different story from that of the average case of Pott's fracture, because this is not an average case of Pott's fracture. It is more than a Pott's fracture. How did she fall?

Intern: She does not remember exactly. All she knows is that she fell on the right foot.

Dr. Murphy: Which way did the foot turn?

Intern: It turned inward and backward.

Dr. Murphy: That tells the story. *The foot turned inward and backward.* You never get a typical Pott's fracture from an inversion trauma. That is where the fundamental error was made, both in diagnosis and in treatment, and that is what led to this disaster. The history alone should have told the physician what was the matter. When the doctor first saw the case he could scarcely make out anything at all as to what the conditions present were; but the story should have told him that there was something else there other than a Pott's fracture, because the fracture was caused by inversion.

Let us see what happened? There was a fracture of the external malleolus on a level with the articular surface of the tibia. It was a fracture of the inner third of the articular surface of the tibia, extending upward a distance of two inches, and carrying inward all the inner portion of the lower end of the tibia. If that had been a Pott's fracture, it would have taken off only the tip of the internal malleolus, and if it fractured the tibia at all, it would have fractured its outer half and carried that backward or upward. The trauma in a Pott's fracture never fractures the inner half of the tibia unless it breaks both bones.

The treatment pursued was the typical treatment for Pott's fracture, and thereby resulted this disaster. The typical treat-

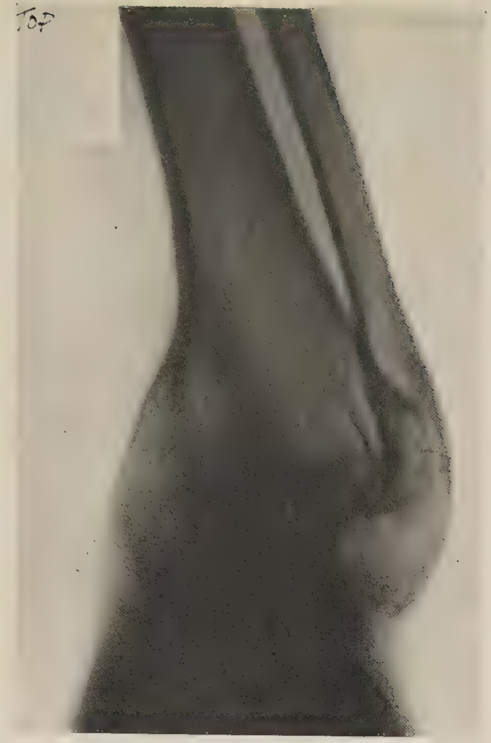


Fig. 273.—Inversion fracture of the right ankle treated as a Pott's fracture with the usual adduction dressing. Consequent development of ankylosis and a severe adduction deformity of the ankle, which is clearly shown in this antero-posterior radiogram made before operation and about two years after the original injury. Note the marked inward displacement with which both fractured malleoli have healed, and the oblique angle at which the line of the joint stands, whereas its position should be horizontal. Note the variance in direction between the perpendicular axis of the tibia and the perpendicular axis of the foot. Note the absence of a joint interval, indicating that the ankylosis is bony.

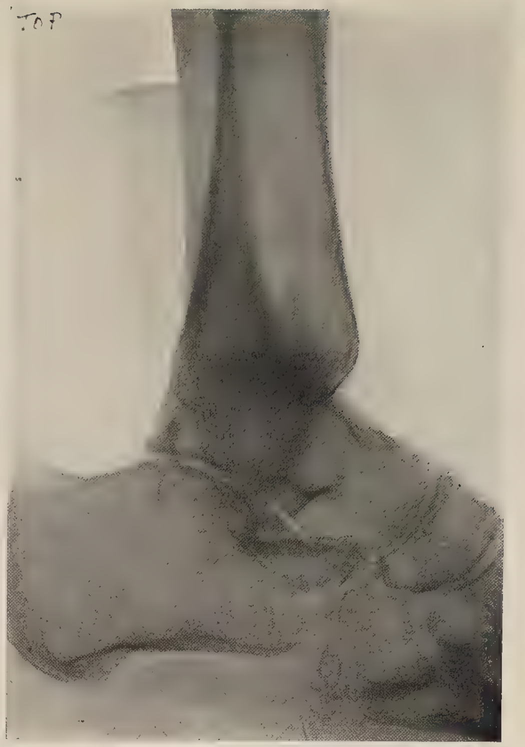


Fig. 274.—Same case. Lateral radiogram made before operation. Note the obliteration of the tibio-astragaloid joint, indicating the presence of bony ankylosis.

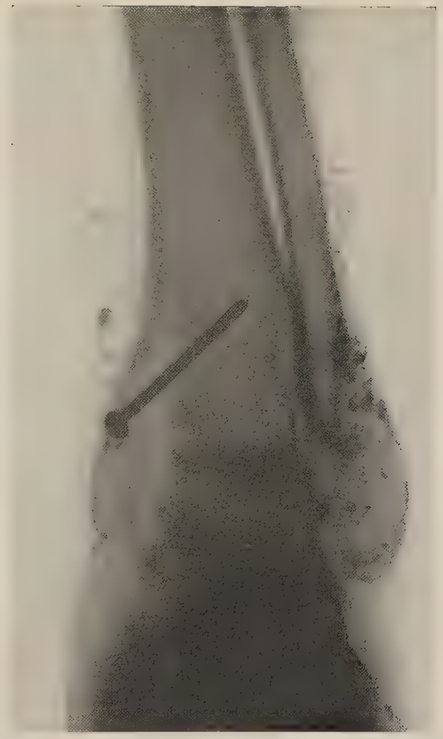


Fig. 275.—Same case. Anteroposterior radiogram made five weeks after operation—open reduction of the deformity. Both malleoli were divided with the chisel at the old line of fracture. The bony ankylosis at the tibio-astragaloid joint was divided partially with the chisel and then broken across by manual force. The internal malleolus was fastened in the corrected position by a wire nail driven through it and up into the diaphysis of the tibia. The external malleolus then remained in good position without further fixation. Note the restoration to the horizontal of the line of the ankle-joint. Note that the vertical axis of the tibia now coincides again with the vertical axis of the foot. Note that bony union has already taken place.

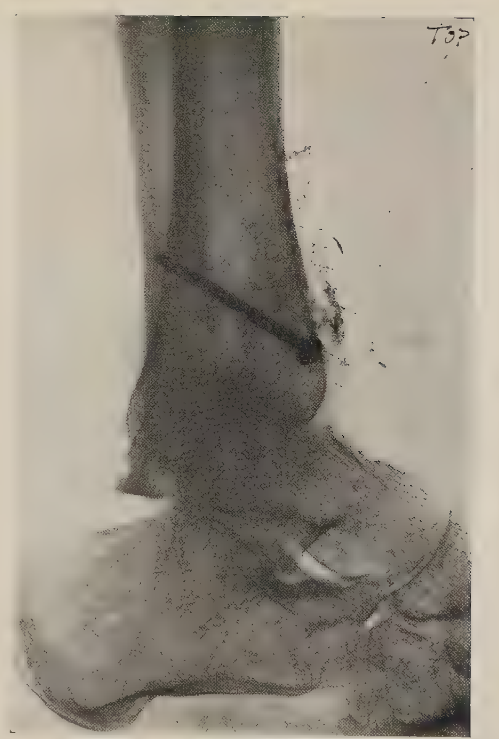


Fig. 276.—Same case. Lateral radiogram made five weeks after the open reduction of the deformity. Note the solid bony union of both malleoli and the free formation of callus in spite of the presence of a foreign body (the nail) and in spite of the rigid immobilization obtained. Rigid immobilization does not hinder callus formation in joint fractures to any great extent, as a rule, whereas in fractures of the shafts of long bones it has a markedly deleterious action on callus formation.

ment for a Pott's fracture is to dress the foot as much inverted and as much adducted as possible, with the foot at an acute angle with the leg. In dressing this foot in superlative adduction the foot and the external malleolus have been pulled halfway across the articular surface of the tibia and the internal malleolus has been dislocated far inward. The inward displacement of the lower fragment of the tibia is possibly one-half to three-fourths of an inch in extent.

I was unable to determine definitely, when she was awake, whether or not she has a complete bony ankylosis of the ankle-joint. Judging from the *x*-ray picture, I believe she has, and, therefore, while I could promise her a straighter limb, I could not promise a movable joint.

Apparently all the internal malleolus must be removed in order to bring the foot perfectly plumb with the leg. This is a very severe deformity. I am sorry that we have no photograph of it, because a good photograph would be valuable in teaching how to prevent such a catastrophe in other similar cases. A clinic is like a mine—it is the day when one expects only the usual grubbing that the real treasure is found. The photograph would not help us fill the present order, but would be of great value for future delivery.

All the pressure in walking comes on the outer side of the foot. That causes her great pain and discomfort. [Internal longitudinal incision.] We shall expose the fracture by making our incision to the inner side of the tendon of the tibialis anticus. By dissecting downward we expose the line of fracture and the ankle-joint. My previous opinion, based largely on the *x*-ray plate, that the ankle is completely ankylosed, is fully verified. There is no motion at all in it. It is perfectly solid. That will make our task much more difficult than it would have been with a few fibrous adhesions and some motion still present. The ankylosis is already of two years' standing.

We shall try to preserve the internal malleolus which we have freed, but must excise the piece of bone which lies between the malleolus and the ankle-joint. That is the new growth of cancellated bone which has filled in the gap produced by the fracture. What little motion we are able to elicit by flexing the ankle is all in the tarsal articulation below the ankle. As soon as all the fragments of new bone are removed we shall be able to bring the foot around into proper position. The bony fixation of the ankle is between the tibia and the astragalus.

In attacking the fibular side of the fracture we make an external longitudinal incision and approach the joint by passing beneath the peroneal group of tendons. The upper end of the lower fragment of the fractured fibula was displaced inward, apparently, on to the articular surface of the tibia. The fibula, which has united in spite of the displacement, must be refractured on a level with the articular surface of the tibia, in order to let the upper end of the lower fragment slip outward over the articular surface of the tibia and come back into proper relation with the upper fragment.

We next have to separate the astragalus from the tibia, to which it is united by a bony ankylosis. [The separation is made first by outlining it with the chisel and then breaking the bones free manually.] Now that the bones and the articulation have been freed, the foot can be placed in any desired position. It must be placed in slight overcorrection; that is, with the foot turned out a little farther than straight.

The deformity having been reduced, it is necessary to hold the bones in the corrected position. For this purpose we shall perform the usual extra-articular nailing of the fragments. We are driving one wire nail through the internal malleolus up into the tibia, and another through the lower fragment of the fibula, including the external malleolus, into both the upper fragment of the fibula and the tibia. The nails will hold the fragments rigidly in position while we are applying the dressings and the wire-gauze splint.

The constrictor was not applied tightly enough to the limb at the beginning of the operation. The arterial circulation was thus allowed to continue while the venous circulation was blocked. Therefore there was more bleeding than if there had been no constrictor on at all. The bleeding practically all stopped as soon as the constrictor was removed.

Let the record show that we had much difficulty in freeing the astragalus from the tibia because of the complete bony ankylosis, of which we informed the patient before the operation; that there will be, in all probability, a return of the bony ankylosis and a complete fixation of the joint, as we warned the patient; but that we hope to have the foot comparatively straight and in line with the leg; that at a second operation we may have to remove some of the prominent portion of the external malleolus, but that it must be kept for the present because of the support which it gives to the joint; that after the completion of the work there was good apposition of the bony surfaces and the foot was apparently plumb with the tibia; that after freeing the astragalus on both sides we succeeded in luxating it outward beyond this plumb line—that is, we overcorrected the previous deformity of the foot.

The pressure on the circulation in the ankle will be considerable when the edema and swelling develop in these tissues, as they always do after such an amount of traumatizing. Have we been able in all the manipulating to keep the field sterile? We hope so. At least we have not touched even our gloved hands to the wound—only metal instruments, sterilized by boiling, and dry gauze, sterilized by steam and hot, dry air.

In every one of these inversion fractures of the ankle we have the same difficulty in reducing the deformity and in securing an approximation of the fragments. We have another danger still to fear, and that is the increased tension on the circulation when this ankle commences to swell. We had one of these cases in which it looked as though the patient was going to lose his foot. A portion of the great toe and the distal joint of the second toe finally sloughed, but still the rest of the foot lived. In straightening this patient's foot around into proper position the tension became so great on the posterior tibial artery that the circulation was shut off. When the swelling developed, the foot turned black; but gangrene, *in toto*, although imminent, was, happily, averted.

Let the record show that on completion of the operation there was practically no tension on the skin-flap, so that its circulation is not hazarded; that we had to use no deep ligatures, thus showing that none of the deeper and important vessels were injured—neither the anterior tibial nor the posterior tibial vessels.

The wound is dressed with gauze moistened with a solution of 1 per cent. carbolic acid in alcohol. The gauze dressing takes up all the wound secretions and blood which escape and prevents them from putrefying. A posterior wire-gauze splint is then applied to hold the dressings in place and the foot in its corrected position, thus preventing any excess of strain on the wire nails.

[Note.—The wound healed by complete primary union except for a small spot of necrosis over the external nail. The latter was accordingly removed, June 30, 1914. The patient is still in bed at the present time, July 6, 1914.—Ed.]

INVERSION FRACTURE OF THE ANKLE TREATED AS A POTT'S FRACTURE BY AN ADDUCTION DRESSING.—CONSEQUENT ADDUCTION DEFORMITY.—OPERATIVE CORRECTION AND NAILING OF THE FRAGMENTS

HISTORY

The patient, a young man aged twenty-four years, entered the hospital November 25, 1913, because of a bony deformity of the right ankle, slight inversion of the foot, and pain in the ankle after walking a considerable distance.

The patient's present trouble dates back to November 1, 1912, when he was thrown violently to the ground while attempting to stop a runaway horse and wagon. The wagon-wheel ran over the patient's right ankle, across its inner side, the outer side lying against the pavement. He was immediately taken to a hospital, the ankle was pronounced broken, the foot was manipulated without an anesthetic, and the fracture was thought to have been put back into place. There was considerable pain in the ankle during the ensuing days, and it very soon became swollen and discolored. On the third day after the accident an *x*-ray picture was taken and the patient was told that the condition of the ankle was "satisfactory." On the tenth day after the injury, the swelling about the ankle having greatly diminished, a circular plaster-of-Paris cast was applied, extending from the base of the toes to about the middle of the thigh. This cast was left on for three weeks, and during this time the patient remained in bed. After the cast was removed the patient got up on crutches, but did not try to walk on the foot unassisted because of the pain caused by any attempt to use it. After using crutches a couple of months, during which time he gradually began to put weight on the leg, he was able to walk fairly well, but always had some pain in the ankle while doing so. Gradually he noticed that he was walking more and more on the outer side of the foot, which, in the course of time, became turned somewhat inward. After this observation he used a cane in walking, and has continued to do so up to the present time. He has had 10 or 12 *x*-ray pictures taken to determine, if possible, the cause of the disability and its course, whether for better or worse. For a considerable time he wore a board-splint strapped on the inner side of his leg, in order to overcome the inversion of the foot. He was told by doctors that the cause of his disability was a considerable amount of callus at the site of the fracture, which in the course of time would be absorbed.

COMMENTS AND OPERATION

DR. MURPHY (November 28, 1913): Now we have this mighty unpleasant case to handle. The doctor who originally treated the case evidently thought that it was an eversion fracture—a Pott's—and so treated it, whereas in reality it was an inversion fracture. The break was more complicated than an ordinary Pott's fracture. Besides the transverse fracture of the external malleolus at the level of the articular surface, there was also an oblique fracture of the lower end of the tibia, involving the inner four-fifths of the lower articular surface of the tibia and extending upward and inward for two and a half inches, as well as a fracture of both malleoli. The *x*-ray shows, too, that the upper border of the astragalus is not level. This fracture was dressed in the direction in which a typical Pott's fracture should be dressed, namely, in superlative adduction, and that was the cause of the subsequent deformity; for this was not a Pott's fracture but an inversion fracture, and should not have been treated as a typical Pott's. As the result of the adduction dressing, the internal malleolar fragment was displaced inward and upward along the inner side of the tibia; and the lower articular surface of the tibia, which should contact with the astragalus, now runs upward toward the inner side instead of horizontally, partly because there was an oblique

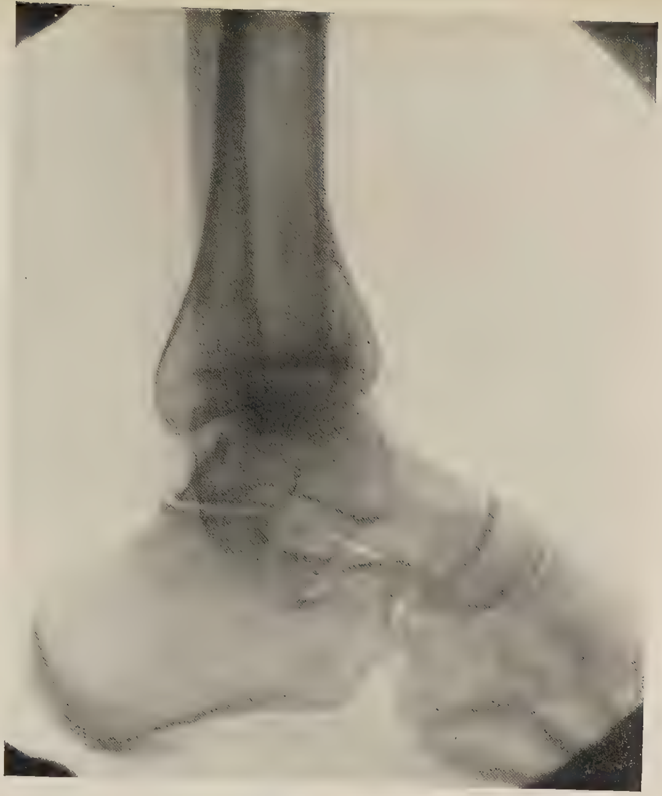


Fig. 277.—Inversion fracture of the ankle treated as a Pott's fracture by an adduction dressing. Consequent adduction deformity. Lateral radiogram before operation. The anterior dislocation of the foot and the slight arthritic changes in the tarsal joints are the most evident features of this picture.

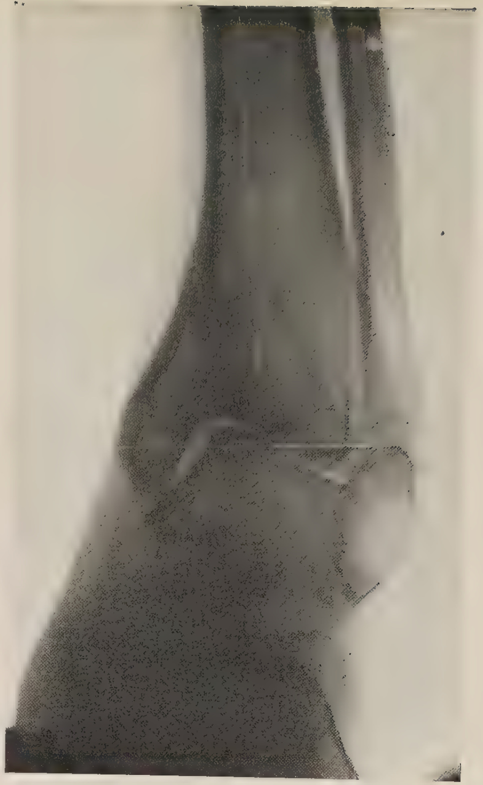


Fig. 278.—Same case. Anteroposterior radiogram before operation. Note the marked abduction deformity at the ankle, the regular and characteristic result of the very common but mistaken practice of dressing an inversion fracture like a Pott's fracture, in a position of maximum adduction. Note the marked adduction of the tip of the external malleolus, the impinging of the internal malleolus on the inner surface of the astragalus, and the oblique angle at which the ankle-joint stands. The two white lines scratched on the plates and crossing each other at right angles at the level of the ankle-joint were scratched on the plate in order to emphasize the marked degree of adduction of the foot and the angular displacement of the line of articulation at the ankle, by thus indicating where the normal lines would lie.

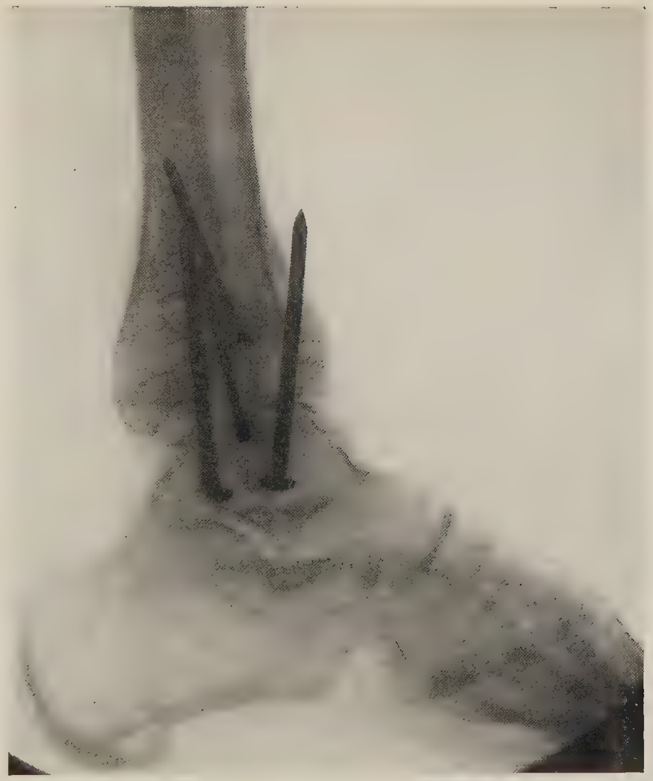


Fig. 279.—Same case. Lateral radiogram made about seven weeks after operation. Comparison with Fig. 277 shows how well the anterior dislocation of the foot has been corrected. Note that the anterior inner nail passes clear through the anterior margin of the tibia out into the soft parts, not the intended goal. Part of the head of the posterior nail through the internal malleolus is missing. It was cast off after the nail was driven in, in order to keep the edge from irritating the under surface of the skin above it.

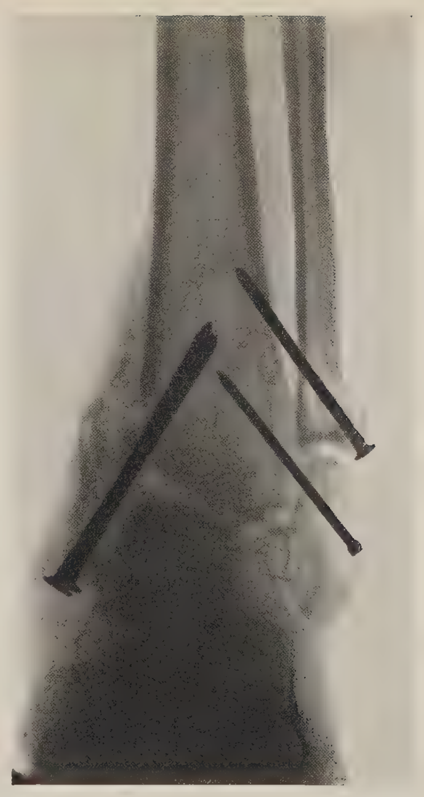


Fig. 280.—Same case. Anteroposterior radiogram made about seven weeks after open correction of the deformity and nailing of the refractured fragments. There is a well-advanced callus formation about the internal malleolus. The refractured fibula shows as yet no sign of the development of a bony callus. The fogging of the outlines of the joint indicates a considerable degree of articular and periarticular reaction following the operation. The tip of the posterior inner nail and the tips of the two outer nails appear to lie in common in a roughly circular area of lessened density in the middle of the lower tibial diaphysis. That this is not an osteomyelitic focus we know from the patient's entire absence of fever. It is probably a lacunar absorption of bone, the result of the presence of the three foreign bodies in it. Note that the nails nowhere invade the joint cavity.

fracture across the lower end of the tibia involving its articular surface, which should have been dressed with the foot straight or in slight eversion. When the ankle was dressed in superlative adduction, there was too much displacement inward of the lower fragment of the fibula, and, consequently, the fragments failed to unite because of the faulty apposition. The patient's ankle is now turned inward and he walks on the outer side of the fifth metatarsal bone. His ankle is still painful and he walks with a limp. His is not an easy case to remedy, and we have had our troubles figuring out a procedure which promises success. I think we must bring the lower tibial fragment downward and outward and fasten it there. We must elevate the external malleolus and reunite it to the fibula, if possible. The internal malleolar fragment must likewise be reunited to the tibia. If one runs a horizontal line through the x-ray picture of this ankle at the level of the joint, one sees well the deviation from the normal. This horizontal line *should* lie parallel with the lower articular surface of the tibia, but, instead, it forms an acute angle with the articular surface because of the faulty position of the joint structures.

We told this man that we probably should not be able to restore flexion in his ankle, but that we could give him a foot which would come plumb with the ground, and would carry his weight securely and without pain. The reason we cannot restore his flexion is because there has been a forward luxation of the astragalus. We probably cannot altogether overcome that luxation, but we hope to relieve him of the pain which it constantly causes him. I stated at the outset that this was not a genuine Pott's fracture, but an *inversion* fracture. Let us see what are the elements of a true Pott's fracture:

First: It is always an abduction lesion, that is, it is always caused by an injury which produces an abduction of the foot.

Second: There is a fracture of the fibula always; and that fracture of the fibula may occur, as it did here, on a level with the articular surface of the tibia, or it may occur anywhere above that level, usually not over two or two and a half inches above, and is usually accompanied by a displacement of the lower fragment.

Third: There is a laceration of the interosseous ligament.

Fourth: There is a fracture of a greater or lesser portion of the tibia, or of the internal malleolus, or of the internal lateral ligament.

These are the anatomic elements of the lesion. Occurring, as it does, as an abduction injury, one readily sees why the fibula breaks first. It is on the fibula that the first strain of abduction comes. The reason the fibula breaks is because the strength of the interosseous ligament is such that it does not permit the fibula to separate from the tibia; and the fibula fractures before the ligament tears. But as soon as the fibula has been broken and the tip of the malleolus displaced outward, the outer upper angle of the astragalus slips up between the fibula and the tibia and splits the interosseous ligament like a wedge, so that there is a laceration of the interosseous ligament always present in an eversion fracture, where the fibula has been broken above the articular surface of the tibia. If the violence of the injury carries the process still further, the internal lateral ligament, the internal malleolus, or even a large portion of the lower end of the tibia may be broken off; and, in injuries of the severest grade, the upper fragment of the tibia may be pushed through the skin, compounding the fracture.

This is a class of cases that we do not particularly like. We often wish we could foist them on some one else. They are not nice cases, as a rule, either to treat or to meet in the years after treatment. They usually have the hammer out for their first doctor, and may love their second no better. We have one such patient who suffered a gangrene of the foot from an operation of this kind. The gangrene was only partial, but he lost the terminal joint of his great toe. He was suffering from an old backward luxation of the foot on the ankle following a transverse fracture of the posterior lip of the tibia. When we brought the foot back into place the tension on the posterior tibial artery was so great that the circulation in the foot was shut off.

[Holds up the patient's foot toward the clinic.]

See the curve that this ankle now has (marked inversion)! The foot was dressed in too great inversion. That deformity cannot occur after an ordinary Pott's fracture no matter what the dressing. [Makes a longitudinal incision over the external malleolus and completes the exposure of the site of fracture by dissection.] Here is the enormous callus which has formed as the result of the fracture of the fibula. There is the articular surface of the lower end of the tibia. Here is the former line of fracture of the fibula, almost on a level with the articular surface of the ankle. With a curved chisel we are cutting away the excessive fibular callus and freeing the external malleolus. Now that it is freed we must expose the internal malleolus. This is the difficult one to manage. [Makes a longitudinal incision over the internal malleolus and frees the malleolus by dissection.] This is a much harder proposition than the management of the external malleolus. When one first looks at that internal malleolus, the reduction of its displacement appears to be an easy proposition to handle; but in reality it is far from easy. If this were merely a simple fracture of the internal malleolus, the problem would not be difficult. It is the combination of the fracture of the malleolus with a fracture of the articular surface of the tibia which makes our task a serious one. We are opening on to the articular surface of the tibia. We must take out enough of that tibial callus to let the internal malleolus and its attached articular fragment come downward and outward into place. [Uses the Jones wrench to swing the foot outward.] Now we have it and are masters of the situation. Without that Jones instrument we should have had very serious difficulty in bringing the fragments into position. We shall now insert the nails. [Drives two wire nails through the internal malleolus up into the shaft of the tibia, another through the lower end of the upper fibular fragment into the tibia, and a fourth through the lower fibular fragment into the tibia.] I will just cut off the head of this nail (the posterior nail driven through the internal malleolus) from the outside, so that I can drive it a little further into the bone and keep it from becoming exposed through the skin incision.

That was a difficult situation to handle. We refractured the external malleolus exactly where it was fractured before; but we reset it at the correct angle. It stayed in position of itself just where it was. We inserted the wire nails, nevertheless, to insure the permanency of our result.

Let the record show that both the malleoli and the articular surface of the joint were accurately exposed; that the bones were refractured and the fragments restored to the normal position; that, in fact, we overcorrected the deformity by producing some hyperabduction of the foot, which was accomplished with the aid of the Jones wrench.

[The skin incisions were then closed with continuous sutures of horsehair after the soft tissues and ligaments had been united with catgut to take the tension entirely off the skin.]

We were masters of that situation once we had the tibia correctly refractured. You can see that we are going to have a favorable outcome. This man will get a much better result than we promised him if he gets through without an infection, as we hope. That will be a nice outcome of an ugly condition. [A copious dressing and a circular plaster-of-Paris bandage over it were then applied. The plaster cast was cut lengthwise up its anterior surface.]

Notice that we aim never to put on a completely circular bandage or cast. If we do use a solid circular dressing, we always split it along one side before sending the patient back to bed. We have a well-founded fear of ischemic paralysis.

Let the record show that we first freed and exposed the line of fracture of the external malleolus; that we then divided the external malleolus, making the incision crescent-shaped with the round chisel, so that the lower fragment of the divided fibula could be rotated outward or inward, as necessary; that after the division of the external malleolus was completed we refractured the internal malleolus and the external line of tibial fracture, extending to the articular surface; that we then excised a V-shaped piece from the tibia at the upper end of the internal malleolus, which included the malleolus and a little more, and that division permitted the malleolus to come downward and outward into proper position; that we put on the Jones wrench and swung the foot outward, bringing the external surface of the astragalus into articulation with the inner surface of the fibula, so that the

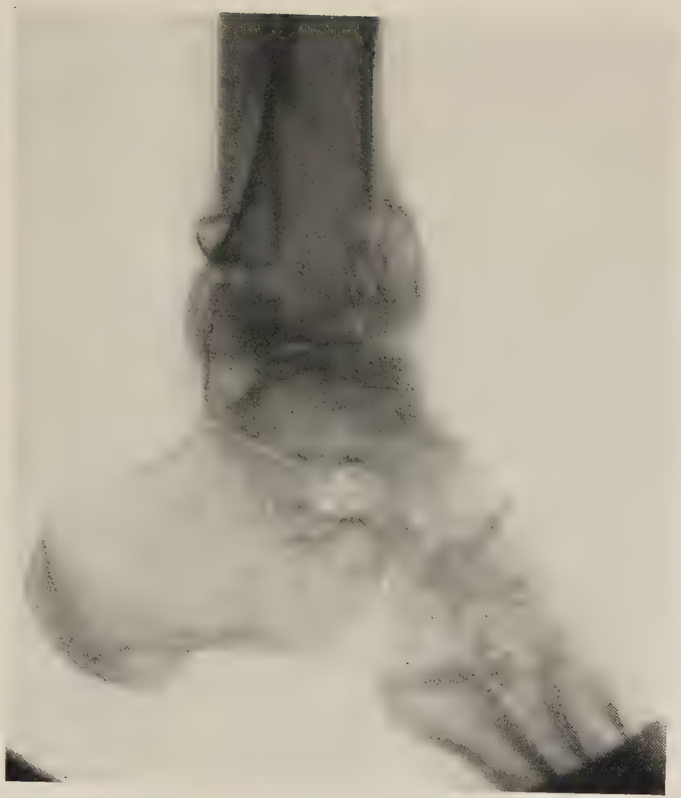


Fig. 281.—Same case. Lateral radiogram made about nine weeks after the original operation and two weeks after removal of the nails. The focus of rarefaction in the lower diaphysis of the tibia does not show clearly in either of the lateral views. Apparently, therefore, its greatest diameter is anteroposterior and is partly overlapped by the shadow of the fibula. The numerous white spots which one sees are not the signs of the spotty variety of bone atrophy, but are defects in the emulsion of the *x*-ray plate used.

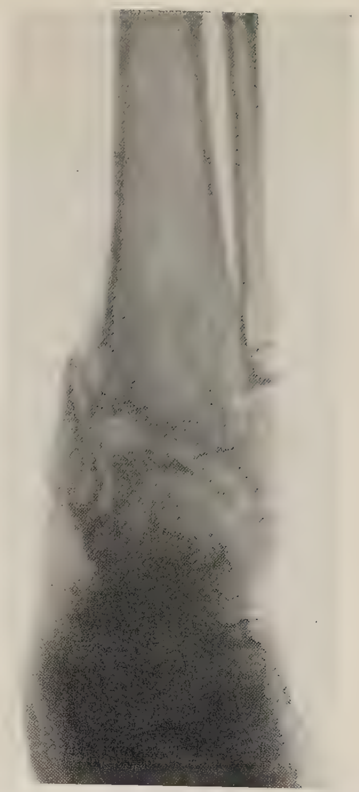


Fig. 282.—Same case. Anteroposterior radiogram made about nine weeks after the original operation and two weeks after removal of the nails. The focus of diminished density in the lower tibial diaphysis is still evident. The external malleolus still shows no sign of bony union. The inner malleolus shows a well-developed mass of callus about it, not yet completely calcified. The alinement of the bones of the foot with those of the leg appears to be perfectly plumb.

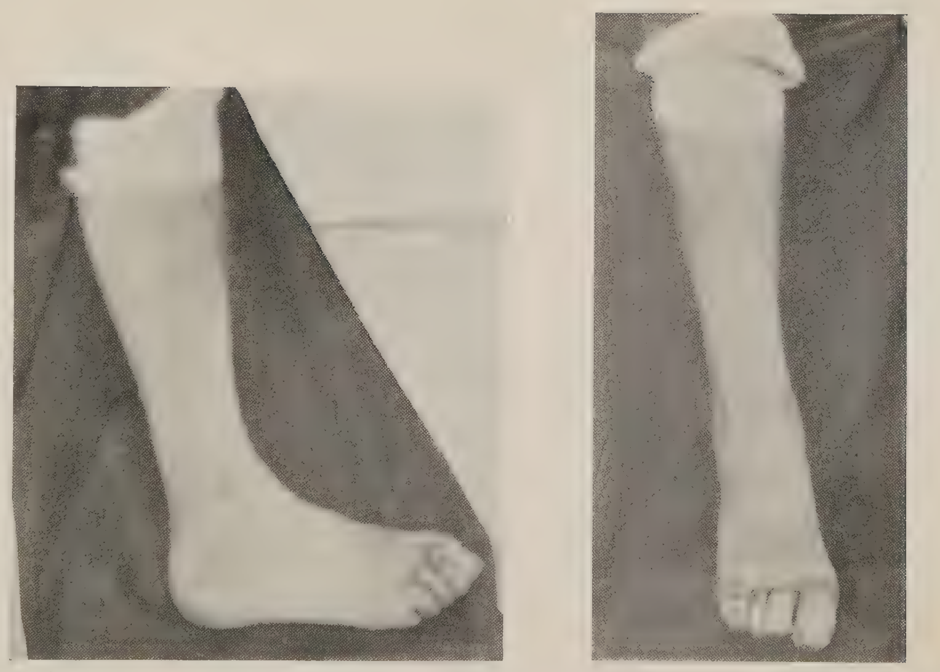


Fig. 283.—Same case. Photographs taken thirteen weeks after the original operation and six weeks after removal of the nails. Note the excellent contour of foot and ankle and the nearly normal range of flexion and extension at the ankle. Note the entire absence of any trace of remaining adduction deformity.

foot remained in slight eversion a little overcorrected; that after the forcible correction of the deformity two nails were placed on each side of the ankle to hold the fragments in position; that the limb was then as perfect in conformation as a normal limb—perfectly straight except for the slight eversion of overcorrection.

We shall elevate this foot immediately on the patient's return to bed; otherwise edema of the foot is likely to develop very quickly.

REMOVAL OF THE NAILS

DR. MURPHY (January 15, 1914): We are going to take out the nails today because they have irritated the skin somewhat and can now be felt through the skin. [Through two small openings, one on each side of the ankle, the four nails were removed. A little thick material trickled slowly out after two of them.] See the discharge which comes out from the site of the nails. It is in all probability perfectly sterile because the patient has had no fever at any time and the wound has remained closed. From now on the patient will not walk as he did when he came to us,—on the outside of the foot,—but, as he should, on the sole of his foot. One never has the kind of a gait which this patient had following a Pott's fracture. That was the gait of an inversion fracture which, because of a surgeon's mistaken diagnosis, was dressed in inversion. Look at the conformation of that foot. See how perfectly plumb it is with the limb. There is a little granulation tissue around the nails. That is the reaction of the tissues to a foreign body and occurs irrespective of its location. The tissues loosen up the foreign body and also soften it, when softening is possible. When it is not exposed, if it be aseptic, its presence is a matter of relative indifference if not too large. We are not closing the nail wounds entirely, you notice. We are leaving them open where the heads of the nails lay.

Let the record show that two nails were removed from the outer side and two from the inner side of the joint. Let the record further show that after the removal of the two nails from the outer side of the joint there was some escape of a pus-like material, notwithstanding the fact that this man at no time has had any temperature; that after the removal of the posterior nail from the inner side of the joint there was also an escape of some thick material like pus; that there was a direct communication between the posterior nail on the inner side and the two nail openings on the outer side of the joint, apparently through the medium of a cavity in the tibial diaphysis (see x-ray plate); that the anterior inner nail appeared not to communicate with the common cavity; that after the removal of the upper outer nail there was an escape of blood rather than of pus.

That statement shows what may occasionally happen with nails as foreign bodies in the tissues. None of those nails communicated with the joint at all, and the joint itself is entirely free from any secondary changes.

[Note (May 5, 1914).—The patient now walks with the limb perfectly plumb. He has from 70 to 80 per cent. of the normal range of flexion and extension in the ankle.—Ed.]

OLD INVERSION FRACTURE OF THE LEFT ANKLE TREATED AS A POTT'S FRACTURE. CONSEQUENT BAD DEFORMITY.—OPEN REDUCTION AND NAILING

HISTORY

The patient, a seventeen-year-old youth, entered the hospital December 14, 1913, complaining of weakness in his left ankle. His present illness dates back two and one-half years, in 1911, when the patient jumped from a height of eight feet, catching his foot in a swing, and fell so that in striking the ground there was produced a marked inversion of the left foot. There was immediate pain in the left ankle, greatly exaggerated on pressure, so that the patient could not walk and had to be carried home. A doctor was called, and he pronounced the injury a fracture of the ankle. The leg was put up in a splint for one week, followed by a plaster cast, which was left on for six weeks with the foot in an adducted position. The cast extended from the toes up the leg to just below the knee. After the cast was applied the patient was allowed to use crutches, but not to bear any weight on the foot. He continued to use crutches for three weeks after the cast was removed. The ankle was then stiff and lame and it was difficult for some time for the patient to walk unassisted.

In the spring of 1912 the patient slipped and again turned his left foot in under him. A doctor treated the injury as a sprain and the patient used crutches for one week.

In the spring of 1913 the patient again sprained his left ankle, the foot turning in and under him. This time he used crutches for two weeks.

Last October (1913), while playing football, the left foot again was turned forcibly in. The patient used crutches for two days because his ankle was too painful to step on. On last Thursday evening (December 11, 1913) his left foot slipped over the edge of the curbstone, as he stepped on it, and he heard something snap as the foot struck the gutter. He fell to his knees. Since then the left ankle has been sore and stiff. On Friday it was swollen.

Examination on Tuesday, December 16, 1913, showed the internal malleolus broken off and displaced upward; the foot lies in inversion.

COMMENTS AND OPERATION

Dr. Murphy (December 17, 1913): Here is a case of a supposed Pott's fracture, which was *not* a Pott's fracture, which is always an eversion fracture, but an *inversion* fracture. It was dressed in the superlative adduction position (proper for a Pott's fracture, but exactly wrong for an inversion fracture), with the regular result—a complete failure. The patient's internal malleolus now lies three-fourths of an inch higher than his external malleolus, because of the displacement caused by the faulty dressing. When the patient walks, his foot constantly turns in. The injury also broke off a piece of bone from the inner portion of the articular surface of the tibia, together with the internal malleolus. What must we do to give this patient a good functional result? We must bring down into proper position that displaced portion of the articular surface of the tibia with the attached malleolus. The left foot in walking always turns in because there is nothing to support the internal surface of the astragalus against the downward thrust of the body on the inner side of the tibia. We must return the malleolus, with the attached tibial articular surface, to its proper place and fix it there securely. The malleolus must come well down along the inner side of the astragalus.

[Dr. Murphy made a longitudinal incision over the inner side of the left ankle along the internal malleolus. Retracting the cut skin edges, the malleolus came into view and was freed from its surrounding soft parts, including the capsule of the joint.]

That is the capsule of the ankle which I am freeing from the

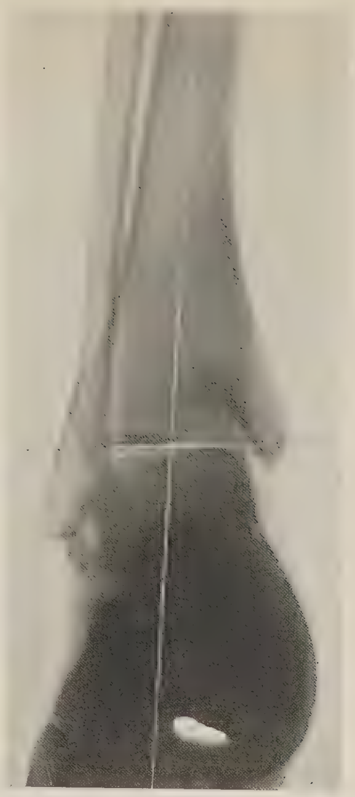


Fig. 284.—Old inversion fracture of left ankle, diagnosed and treated as a Pott's fracture. Consequent upward displacement of the fractured internal malleolus and adjacent articular surface of the tibia. Frequently recurring inversion sprains, the result of the lack of support to the thrust of the body weight at the inner side of the lower tibial articular surface. Note the high position of the tip of the internal malleolus, only a trifle below the upper articular surface of the astragalus. Also note the wedge-shaped defect in the lower articular surface of the tibia, the result of this upward displacement of the malleolus plus bone absorption, due to the faulty dressing of the ankle, which should have been dressed in eversion, not inversion. Anteroposterior radiogram.

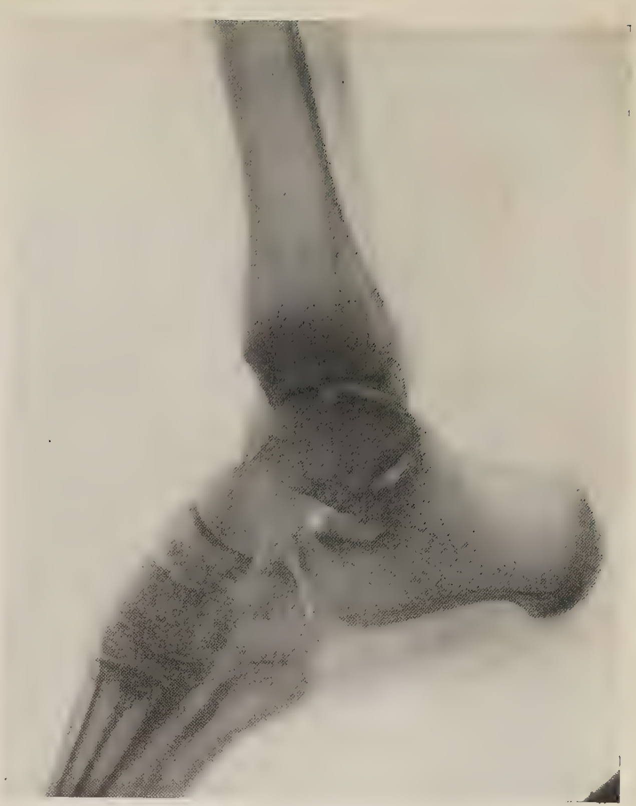


Fig. 285.—Old inversion fracture of the left ankle (lateral view).

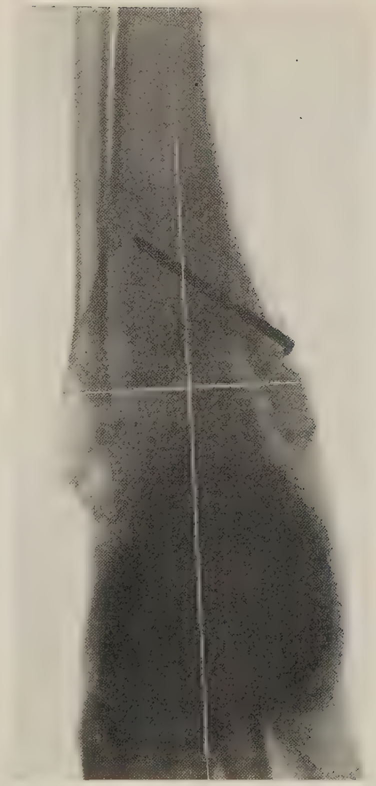


Fig. 286.—Old inversion fracture of left ankle. Anteroposterior view after operation. Note the increased support given the ankle-joint by the lowering of the internal malleolus for three-fourths of an inch, and by evening up, so far as possible, the inner portion of the articular surface of the tibia.

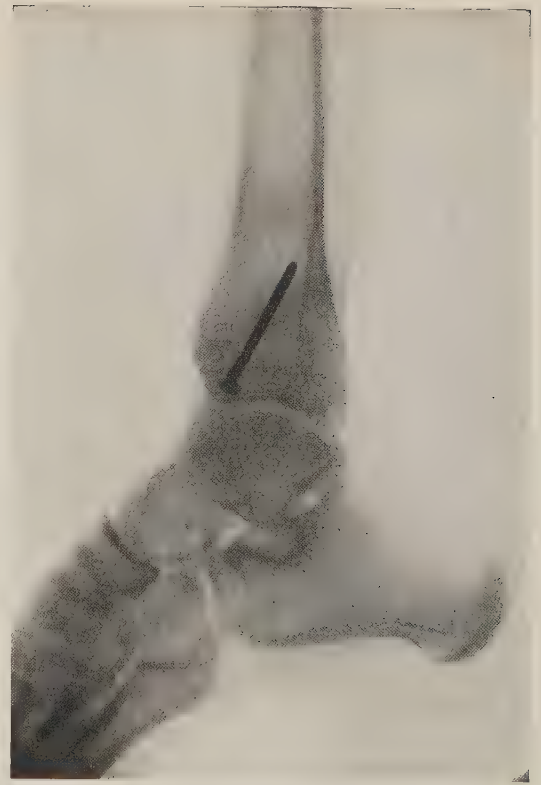


Fig. 287.—Old inversion fracture of the tibia. Lateral view after operation, showing the wire nail holding the downward-displaced malleolus in close and accurate apposition with the tibia, just as the preceding radiogram also shows.

malleolus. The fibrous capsule of the joint comes out over the bone. There is the old line of fracture of the malleolus. Here is the tendon of the tibialis posticus muscle as it comes down behind the malleolus with the tendon of the flexor longus digitorum. One can see from the *x*-ray charts of the circulation about the ankle that the blood-supply to this region is ample. As we are dissecting here, the little spurters seem to come from everywhere. I want the wound kept wiped dry, so that I can get my bearings as to just where the articular surface of the tibia lies. The articular surface of that internal malleolus seems to fit right over the spot for which I am searching. There it is at last. It was much displaced upward.

[Dr. Murphy, with the artist's chisel, refractured the internal malleolus and the attached fragment of the lower articular surface of the tibia, making a V-shaped cut in the inner side of the tibia. He then displaced the malleolus downward for about three-fourths of an inch in the groove thus produced. The internal malleolus, fitting accurately, like a wedge, into this groove, from which it was raised by the chisel, was fastened still more firmly in place by a tenpenny wire nail driven through the malleolus into the shaft of the tibia.]

We shall insert one wire nail to hold the malleolus in the position to which we have brought it. We thus have made a new mortise for the astragalus, so deep that the astragalus cannot slip out on either side.

This case is a fair example of this promising new line of fracture work of ours—the management of fractures near joints by single or double nailing. We are trying to advance and improve this technic and are very much interested in the method just now. Most of these joint fractures we fasten with a single nail, first drilling in the bone a hole just big enough to admit the nail, and then driving in the nail. We avoid compounding the fracture by using only very small skin incisions. This is the practice which all surgeons are coming to, I believe, in the very near future, in the primary treatment of a great percentage of joint fractures, as well as in these late cases. In the discussion of this subject in the Surgical Section of the American Medical Association at Minneapolis last year most of the surgeons who testified stated that they have been getting satisfactory results in the treatment of fractures near joints without the use of this method. All one has to do to convince himself of the desirability of this technic is to stay here for a month or two and see the failures of the ordinary treatment of these cases which come to us for relief. Our success is regular. Good functional results *must* continue to follow in the train of good anatomic results.

VISITING DOCTOR: Did the nail lacerate the articular surface anywhere during its insertion?

DR. MURPHY: No, the nail did not go through the articular surface at all. Our extra-articular, non-bloody nailing of fractures firmly fixes the fragments in pretty nearly every case without compounding the fracture or opening the joint. I am now having a "drill-nail" and "drill-screw" made, which I can insert into the bone as a drill and then clip off flush with the surface when it has been driven in far enough to hold the fragments together. This "drill-nail" shortens and simplifies the procedure, and makes success even a little more certain than with the wire nail.

Let the record show that with the chisel we freed the internal malleolus well over on to the articular surface of the tibia and for a distance of one and three-fourths inches above the articular surface, cutting out with the artist's chisel a V-shaped piece of bone, together with the internal malleolus; that we then displaced the malleolus downward and chipped off the protruding inner part of this fragment at the articular level of the tibia where it was highest; that we then straightened this inner malleolar fragment back into position in the groove prepared for it, pushed it downward three-fourths of an inch, and secured it in its new position with a single nail.

[NOTE.—The wound healed by complete primary union. The patient went home at the end of about six weeks on crutches. No further report to date (September 10, 1914).—ED.]

OLD POTT'S FRACTURE.—INTERNAL MALLEOLUS BROKEN OFF AND ROTATED, PRODUCING NON-UNION.—OPEN REDUCTION AND NAILING

HISTORY

THE patient, a woman aged twenty-nine years, entered the hospital with the following history: On Christmas Eve, 1913, she went riding in a buggy. The horse became frightened and ran away. The buggy was overturned, throwing the patient out. She struck on her right foot. When she tried to rise she noticed that she could not stand on or move her right foot, over which she had lost control. She does not know whether the buggy ran over the limb or not. She does not remember whether the foot was inverted or everted by the accident. She was taken to a hospital and the foot put in a splint for ten days with an ice-bag over the ankle. On January 1, 1914, the foot and leg were put in a cast and kept there for six weeks. She began walking with the aid of a crutch about March 1st, two weeks after the cast was removed; but it was painful to bear weight on the right foot, and, therefore, since then she has worn a leg-brace. There is also eversion of the foot when she steps on it without the brace. There is not much pain at present.

COMMENTS AND OPERATION

DR. MURPHY (June 9, 1914): Here is an *x*-ray which is instructive. (See Figs. 288 and 289.) I think this is the second case of its kind that I have had. They are the only two cases I know of. One I had many years ago—away back in 1886—in a young man. The case was of very much importance to me, for the man was of importance to the world at large. His horse ran away and he was thrown out of the wagon. He suffered a Pott's fracture, with a separation and rotation of the internal malleolus and a consequent failure of union.

Now, then, here is just such another case which has come into our hands. This patient, too, had a Pott's fracture. It was dressed in the usual way by one of our Chicago colleagues, with perfect adduction and turning in of the foot. As a sequence of that dressing she has obtained a splendid union of the fibula with only a slight bowing in. She has obtained an exceptionally good union of her tibiofibular ligament, which is the all-important factor in obtaining a good functional result after a Pott's fracture; but there is a failure of union with a rotation of the internal malleolus. You can see it beautifully in that *x*-ray picture taken of the foot dressed in the classic way. The dressing produced an almost perfect result in every particular over which the doctor had control. Over the matter of union, however, we have practically no control beyond the simple and well-known measures which favor but do not make certain bony fusion. Whether the bone fragments reunite or not when they are placed in apposition with one another is a matter with which the doctor usually has little or nothing to do. All he *can* do with certainty is to make the apposition correct. He cannot greatly influence bone metabolism. We know from the surgical teachings of the last two years and from our clinical records here of many fractures dressed by us and by others that with the very best coaptation and the very best apposition of the fragments there will occasionally be an absence of osteogenesis in the injured bone. That is one of the considerations which has led me in the recent numbers of the CLINICS to write so much about the necessity of avoiding too firm a fixation of the limb in dressing fractures of the shaft of any of the long bones; that a fracture in the shaft of a long bone, if absolutely fixed by very rigid immobilization, such as a plaster cast, nailing or wiring, or a bone-plate gives, lacks enough stimulation at the broken ends to produce active osteogenesis in them; and that there consequently is a failure of union. That, I believe, is why the mechanical fixation by open operation is playing such an important

Fig. 288.—Old Pott's fracture and non-union of internal malleolus due to its rotation. Anteroposterior radiogram made fifteen weeks after the injury. The fracture of the fibula, three inches above the joint, has healed with some lateral displacement and slight shortening. The fractured internal malleolus rotated, producing non-union and consequent eversion of the foot and disability in walking.

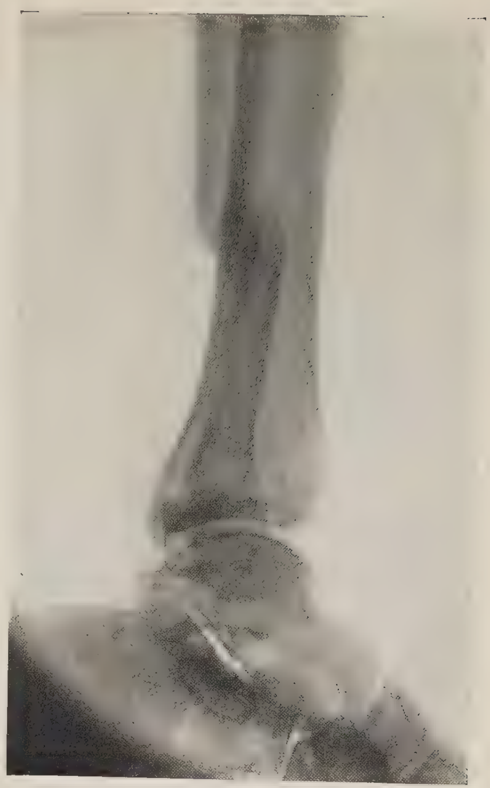


Fig. 289.—Old Pott's fracture with non-union of the internal malleolus due to its rotation. Lateral radiogram made fifteen weeks after the injury. Note that there is present here a slight grade of the "spotty" variety of bone atrophy—an atrophy of disuse.

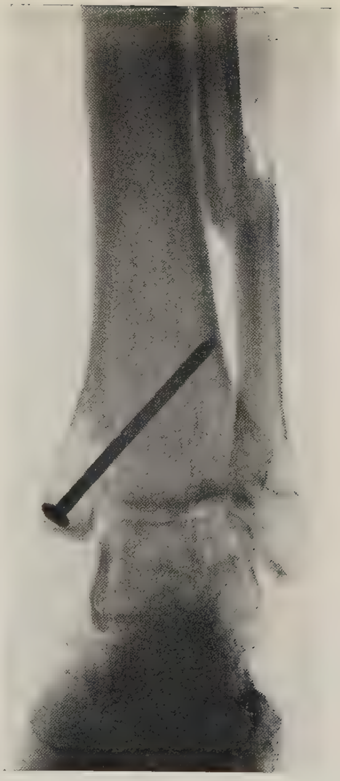


Fig. 290.—Old Pott's fracture with non-union of the internal malleolus due to its rotation. Open reduction and nailing. Note the accurate apposition and solid support thus obtained. The nail is entirely extra-articular. Anteroposterior radiogram made about three weeks after the operation. The spotty atrophy of the bone is a little more accentuated than before operation, as the consequence of the complete immobilization of the ankle.

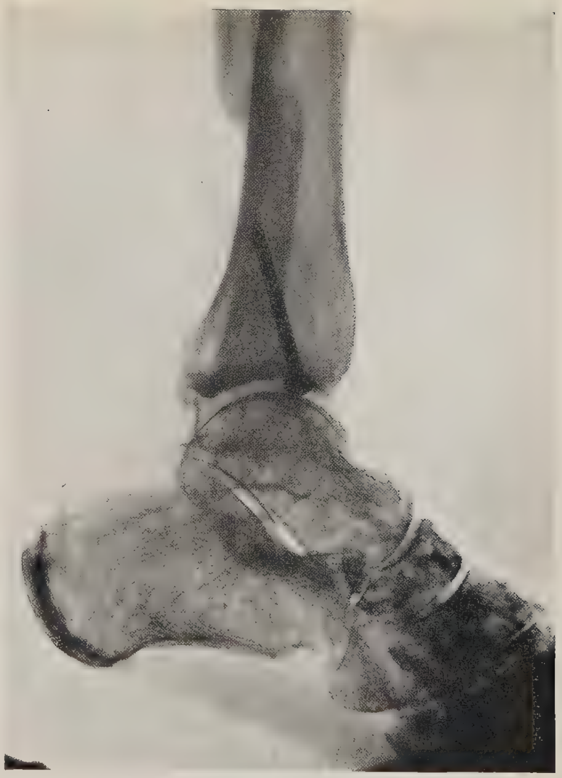


Fig. 291.—Old Pott's fracture. Lateral radiogram made three weeks after the operation. The "spotty" atrophy of the tarsal bones is quite noticeable here.

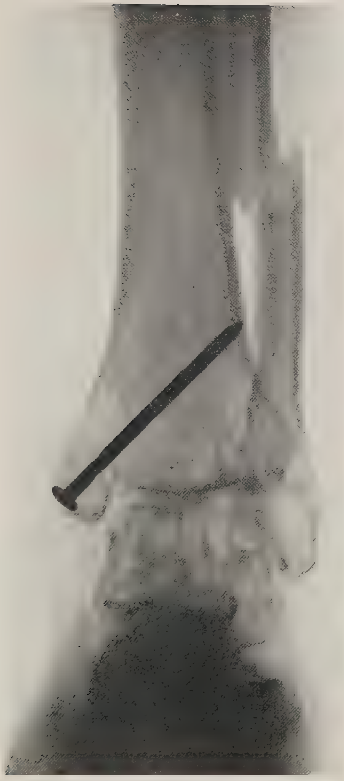


Fig. 292.—Same case. Anteroposterior radiogram made twelve weeks after operation. The nail, still in situ, already shows slight erosion of the shaft of the nail not far from its head and near the old line of fracture of the internal malleolus. This erosion appears to be due to the action of body fluids on the nail, the first step in the process being oxidation, probably.

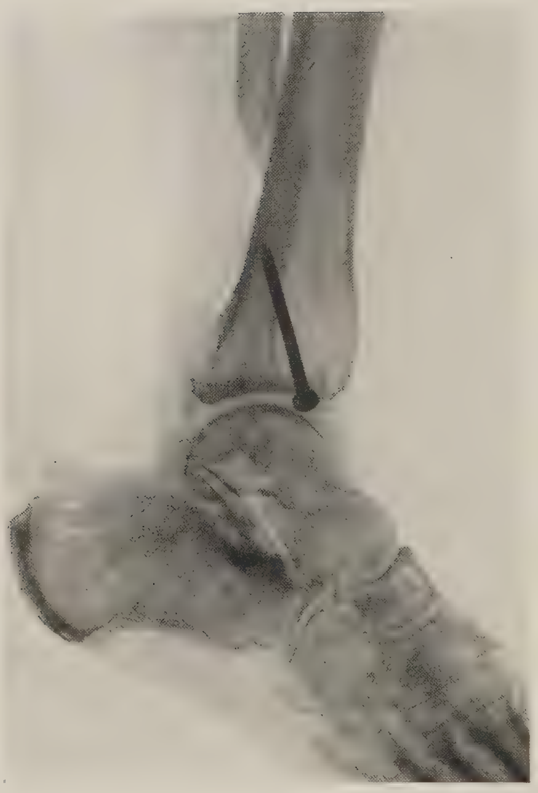


Fig. 293.—Same case. Lateral radiogram made twelve weeks after operation. The bone atrophy, while still present, appears to have undergone some *restitutio ad integram* since the previous radiograms were made.

rôle in producing the present great frequency of post-operative non-unions.

[Dr. Murphy demonstrates the photographs and x-ray plates of another patient. See pp. 977-985 of this issue of the CLINICS.]

Here is another case of a Pott's fracture with a bad result. It was given the typical Pott's dressing in the superlative adduction position; but the fracture was not a typical Pott's: it was a fracture of the internal malleolus. That man got a bad result from his doctor's dressing because, in addition to the breaking off of the internal malleolus, he also had a splitting upward of the tibia. When he came to us he was walking on the outer side of his foot. We got a good result out of it ultimately, as you see, by operative measures.

In this case today the result of the dressing, I should say, is superlatively good, because a plumb line through the center of the tibia would pass through the middle of the astragalus. I think that limb is not one-hundredth part of an inch out of plumb. It is perfectly adjusted. Here in this other case, however [demonstrating again the x-ray plates], the plumb line did not pass through the middle of the astragalus at all. It was thrown away over to the inner side. That was a bad result, for the foot was greatly everted as the consequence of the internal malleolus being broken off. We replaced the malleolus by open operation, put the limb in a plumb line, drove in two nails to fix the bones in the new position, and achieved this splendid result. [Shows patient's photographs.] It is not enough to demonstrate that our performance was mechanically correct; we must show, too, that we produced a serviceable leg for the patient.

This is not a pleasant task which we have before us now. All these operations about the ankle-joint are much more difficult than the similar operations about the knee. Some of the visitors present today may remember the case we operated on Thursday (June 4th), and what a very severe task it was to get that foot around into proper position. The anatomic and functional relationships with which we have to deal in the ankle-joint are more complicated and hence more difficult to manage than in the knee-joint; and the functional stresses which the ankle must withstand are more severe than those put upon the knee.

[Dr. Murphy made a longitudinal incision over the internal malleolus, carrying the cut immediately down to the bone. The edges of the incision were drawn apart by retractors and the soft parts and attachments separated from the internal malleolus and adjacent tibia by blunt dissection.]

There is a line of fibrous union between the separated malleolus and the tibia which I must free so as to be able to rotate the malleolus, and to get it back into proper approximation with the tibia. [Continues dissecting.] Now the malleolus has come around into position beautifully, so that the line of separation is only barely apparent.

[Dr. Murphy then fixed the malleolus in place with an eightpenny wire nail driven through it up into the tibia.]

One can scarcely see the line of division now. The nail extends well into the tibia through the malleolus. [The soft parts were then drawn back over the seat of fracture and the skin wound closed.] I am putting in three tacking stitches of catgut to unite the soft parts and take the tension off the skin, so that there will be no danger of our cutaneous horsehair approximation sutures breaking. We were careful in performing this operation not to touch anything that came into contact with the wound. That is the key to successful bone work. In putting in the buried knots of catgut my gloved hands did not come in contact with the wound, nor did any portion of the catgut that touched my hand come in contact with the wound.

We leave the nail *in situ* indefinitely in most of these cases. Ultimately, we shall probably take out this nail, but not for a long time,—probably three or four months,—by which time the patient will have a firm bony union between the fragments and no longer need the fixation and support which the nail provides.

Let the record show that there was a complete failure of bony union between the internal malleolus and the tibia; that the malleolus had rotated on its longitudinal axis as a pivot, thus turning away its fractured surface from the fractured surface of the tibia.

When this old fracture has firmly united we shall probably elongate and transplant the tendo Achillis so as to rotate the ankle around inward and overcome the existing eversion. That correction should not be attempted now. It is a matter of only a few minutes to carry out, but it requires a solid ankle to withstand the necessary manipulations.

Let the record show that the upper or fracture surface of the internal malleolus had been rotated outward by the traction of the soft parts on it and that I had to free the soft parts in order to let the malleolus turn in; that after freeing it and freshening its broken surface and the adjacent surface of the tibia we succeeded in securing an accurate apposition of the fragments with a single nail. So accurate, in fact, was it that one could scarcely see where the line of separation had been when we were through with its fixation; that there was an eight-penny nail driven up through the malleolar fragment into the shaft of the tibia and fairly well buried under the soft parts; that a light plaster cast was applied; that three catgut stitches were used to reunite the cut soft parts, chiefly connective tissue, before the skin sutures were inserted; that the skin was closed with horsehair.

[NOTE.—The wound healed by complete primary union. The patient left the hospital on July 14, 1914. Her condition was good and there were already firm bony union of the fragments and no swelling of the foot. The motion in the ankle was only slightly limited, and will result eventually in perfect mobility. She was allowed to walk alone on her ankle with the aid of crutches at the end of eleven weeks. She returned to the hospital September 2 for passive motion and *x*-ray pictures. She can now walk without mechanical aid and without pain (September 14, 1914).—ED.]

REMOVAL OF NAIL FROM THE RIGHT TIBIA AND OS CALCIS

[This case, a spontaneous painless inversion fracture of the right ankle in a tabetic patient, followed by the development of a Charcot joint, was originally reported in the February, 1914, Clinics, page 12. The foot was originally dislocated inward to such an extent that the patient's external malleolus almost rested on the ground. The patient could not bear his weight on the foot—not because it was painful, but because it gave way under him. Dr. Murphy reduced the fracture-dislocation and fastened the fragments in their correct position by three wire nails, all inserted by the bloodless method, and only one nail passing through the ankle-joint—that one which was driven through the heel and up through the os calcis into the tibia. After healing had occurred, the upper two nails were removed. An attempt was made at the same time to remove the lower nail,—that driven through the heel,—but the attempt proved unsuccessful because of the great firmness with which the nail was engaged in the bone. More time was, therefore, allowed to elapse, in order to permit lacunar bone absorption to occur around the nail, thus loosening it, before making a second attempt at removal.—Ed.]

Dr. Murphy (June 18, 1914) [Pointing to the *x*-ray plate]: Here is a place where union did not occur between the tibia and os calcis. There is the remaining nail still fastening the tibia over the calcaneus. I went in after that nail once before, at the time I removed the two others, and just could not get it out. It was too firmly embedded. Our nail forceps could not budge it. There is no bony union between the patient's tibia and his astragalus because his astragalus has practically entirely disappeared during the course of his trouble. This is a typical Charcot joint, its internal structure greatly altered, yet never painful, because of the more or less complete regional anesthesia. It is this peculiarity about Charcot joints which leads oftentimes to unexpectedly unfavorable results after treatment of injuries of them or in their vicinity. Such cases often appear to get along very nicely following the injury, and seem to give a good immediate result; and yet the ultimate result may be highly unsatisfactory. In the case of injury to the ankle, for instance, tabetic patients may suffer a Pott's fracture and, after treatment with the typical adduction plaster-of-Paris dressing, may be discharged at the end of five or six weeks apparently completely cured. Then the foot commences gradually to evert or invert, absorption of the bone in pressure areas starts in, and, finally, the patient comes back at the end of three months with a beastly result—a typical, well-advanced Charcot joint; and, too often, he attempts to lay the blame for this outcome at the door of his surgeon.

It is the regional anesthesia or hypesthesia in the lower extremities of these patients which is to blame for the bad result. Faulty dressings, lack of padding, overcorrection, and cramped positions produce no discomfort in the anesthetic joints of tabetics and, therefore, remain uncorrected or are not corrected sufficiently early. Moreover, a tabetic often will walk unconcernedly about on a sprained or even a broken ankle, thus piling Pelion on Ossa in the matter of adding injury to injury. That it is the regional anesthesia which lies back of the development of these Charcot joints rather than vague and mystical "trophic changes," that handy cloak for etiologic ignorance, may be seen from the fact that Charcot joints occur only in anesthetic or hypesthetic areas, in the knees and ankles in tabes, and in the elbows and wrists in syringomyelia. Whoever saw a Charcot joint in a tabetic elbow, wrist, or shoulder unless that patient had a tabes cervicalis rather than a tabes dorsalis?

[Dr. Murphy points again to the *x*-ray plate.]

There is the nail I could not pull out a few weeks ago. Now all I have to do is to open the door, as it were, and let it run out. It is just the same nail. The only change was in the bone. What has happened? An osteoporosis has been set up around

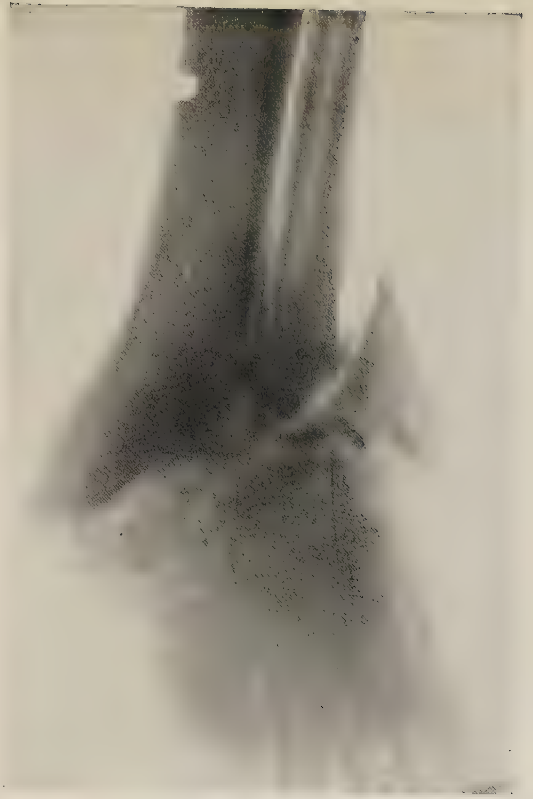


Fig. 294.—Inversion fracture of the ankle in a tabetic erroneously treated as a Pott's fracture. Consequent development of a Charcot joint with a high-grade adduction deformity. Non-union of both malleoli in spite of much erratic development of callus. Nearly complete absorption of the posterior two-thirds of the astragalus. Because of the high-grade adduction deformity and the lowering of level of the ankle-joint by the destruction of the astragalus, the external malleolus came close to the floor in walking. The articular surfaces of the ankle are the tibia above and the os calcis below. Anteroposterior radiogram made shortly before correction of the deformity.

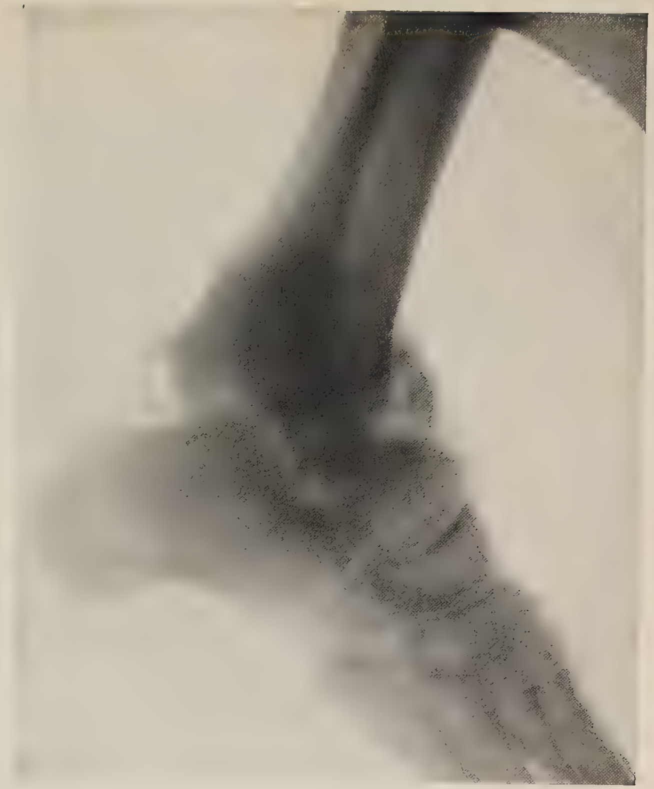


Fig. 295.—Same case. Lateral radiogram made shortly preceding Dr. Murphy's correction of the deformity.

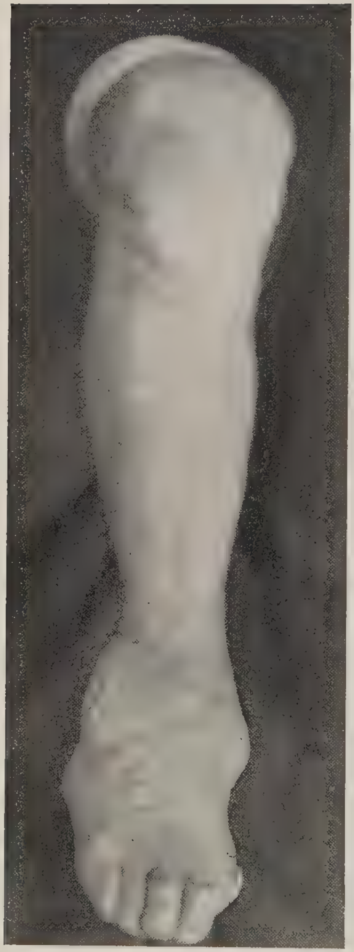


Fig. 296.—Same case before correction.

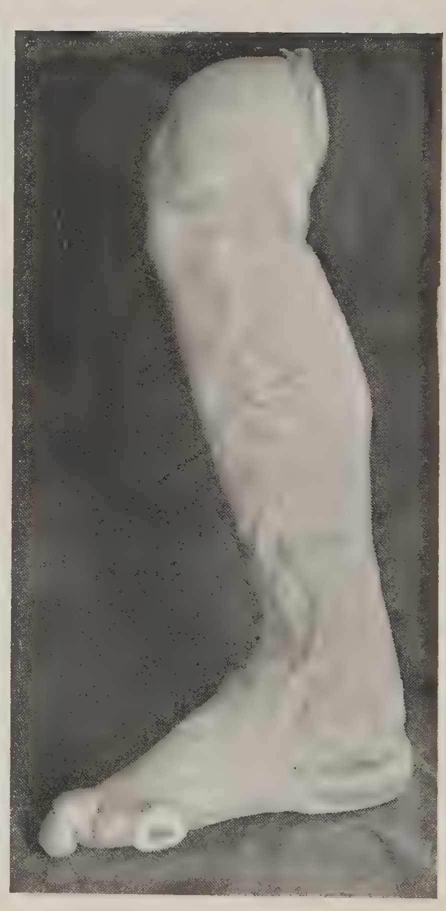


Fig. 297.—Same case before correction.

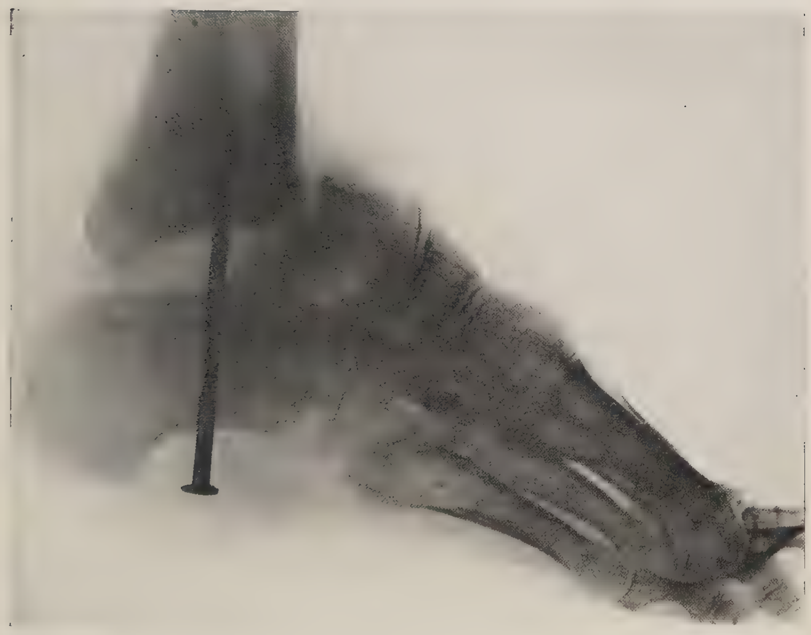


Fig. 298.—Same case. Lateral radiogram made eight and a half months after Dr. Murphy's correction and nailing, and just before extraction of the remaining nail. The two upper nails were removed about four months before this radiogram was made. Note the disappearance of the exuberant callus of the previous radiograms and the clearer outlines to the bones, meaning that less bone regeneration is going on because less bone destruction is taking place now that the previous static deformity has been overcome.

that nail, perhaps with a little hematogenous infection added, months and months after the original operation, when the place seemed entirely healed. [Makes an incision over the head of the nail, grasps it with an artery forceps, and the nail slides out of the heel without effort on Dr. Murphy's part.] That is just what I told you—that this nail was already loose. That shows what happens around a large foreign body long left in the tissue. It must be about four months now since I tried in vain to take out this same nail.

Let the record show that since the previous operation bone absorption has continued in the astragalus until practically none of the articular and posterior portions of the astragalus now is left; that the nail was removed without difficulty and that no pus exuded down along the tract to the surface. The loosening up of the nail, therefore, was due merely to lacunar bone absorption about the nail as about any foreign body long lying in bone, and not to any long latent or recently developed local infection.

[NOTE.—The patient left the hospital immediately after the operation and has not returned since then for observation (September 10, 1914).—ED.]

A RECENT REPORT FROM AN OLD CASE OF KNEE ARTHROPLASTY

Dr. Murphy (June 13, 1914): I had a very pleasant surprise yesterday. A nurse from Sydney, Australia, in coming through California, met a patient of mine who was operated October 28, 1911, for a bony ankylosis of the knee; and the patient sent me these pictures by the nurse (see the accompanying illustrations).

This patient had originally a very bad bony ankylosis of the knee. She was one of the cases in which we established a complete rotation of the patella in performing the arthroplasty in order surely to prevent subsequent recurrence of the ankylosis. We "turned the woolly side in," so to speak, in dealing with the patella. On looking at the *x*-ray plate of the ankylosed knee one sees that the knee was ankylosed at almost a right angle. In "turning the patella turtle" we reversed the aponeurosis of the patella so that it rested against the femur. Here we have, for comparison, the radiograms and photographs before and after operation. This young lady, the nurse, brought me these last pictures, which were made on June 1, 1914. The patient was operated October 28, 1911. The nurse states that she had known the patient for two or three days before she found out that she had had an operation on her knee. If the patient had not told her, she would not have known it. The patient is a very bashful woman. She went out in the alley behind her home to have these last pictures taken by a girl friend rather than go to a professional photographer. They are not the pictures of an artist, but they show well the final result of our operation. In one picture the patient is holding up the operated leg voluntarily without any artificial support. She has the power of flexion to at least a right angle. In this second picture she is standing straight and one can scarcely tell which knee is the operated one, the conformation is so perfect. That, I believe, is one of the chief reasons why we have had good results in this line of work. We endeavor to restore the normal conformation of the joint, so far as possible, in performing the arthroplasty. The patient is about equally knock-kneed in both knees, but walks perfectly. This is a case where the flexible joint is the result of an operative rotation of the patella. Such rotation of the patella insures freedom against return of the ankylosis, because the patellar aponeurosis will not unite to the femur. The operation is technically difficult, except where the ankylosis is at an acute angle. We had a little caries occur at the tip of one patella after turning it in such an operation. We have never had a separation of the ligamentum patellæ or of the quadriceps tendon.

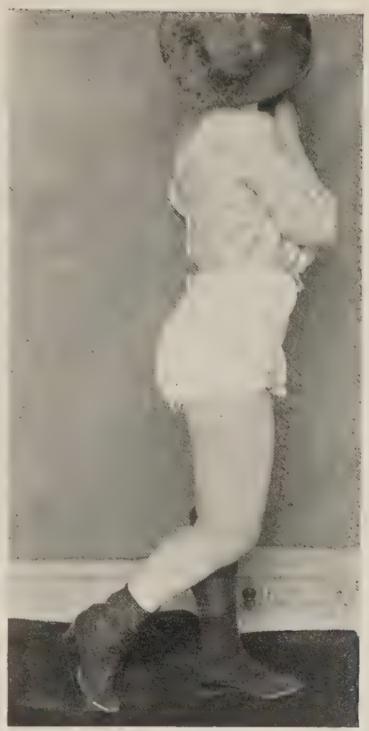


Fig. 299.—Bony ankylosis of the right knee. Photograph before operation, showing the angle of fixation.

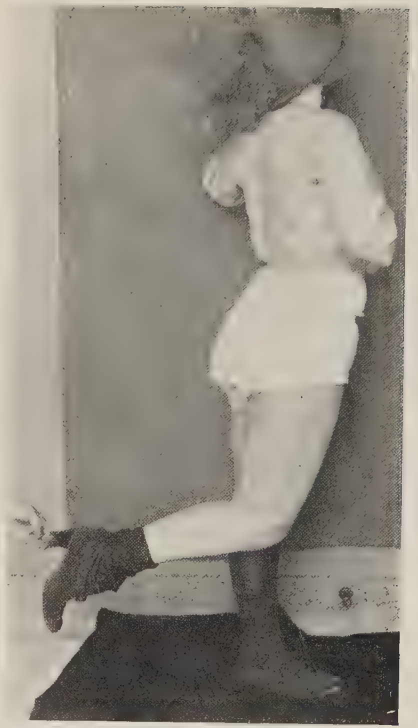


Fig. 300.—Bony ankylosis of the right knee. Photograph before operation, showing the angle of fixation.

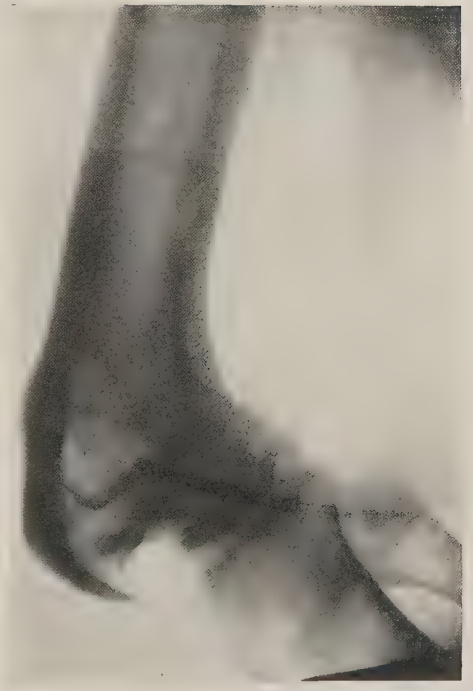


Fig. 301.—Bony ankylosis of the right knee. Patella united with the lower end of the femur. Lateral radiogram before operation.

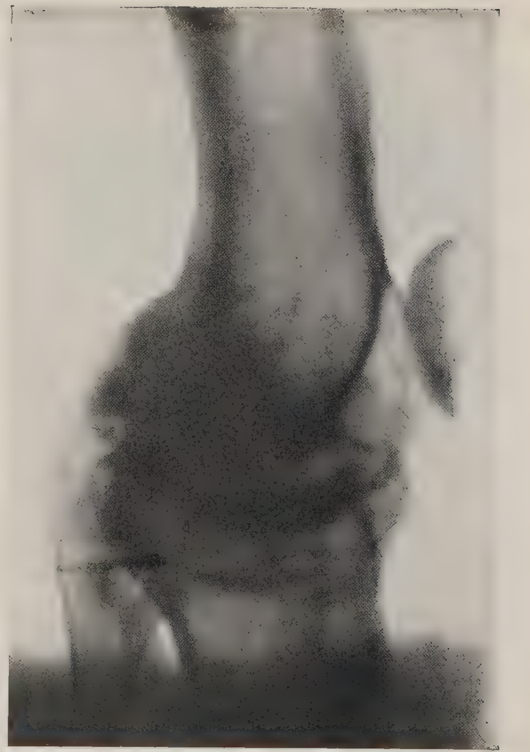


Fig. 302.—Arthroplasty for bony ankylosis of right knee. Lateral radiogram shortly after operation. The patella is seen in profile, "turned turtle," with a free interval, the thickness of the patellar aponeurosis, bursa, and fat, separating it from the femur.

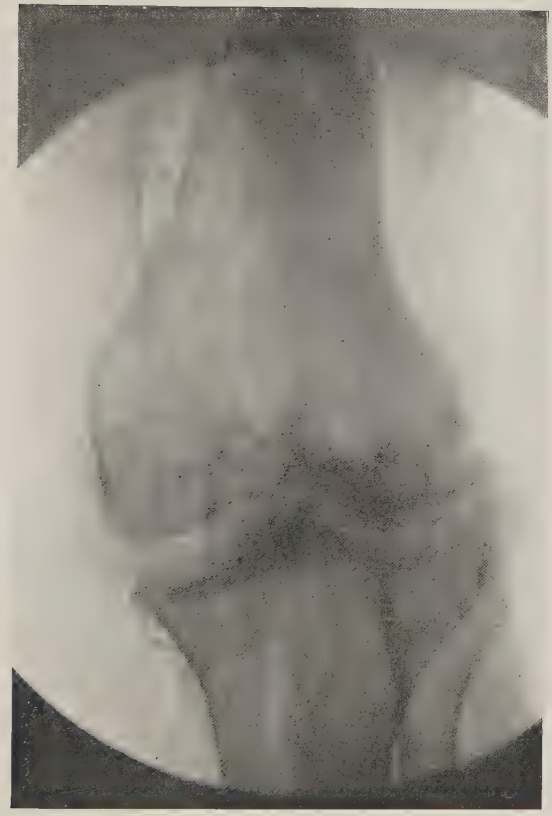


Fig. 303.—Arthroplasty for bony ankylosis of the right knee. Anteroposterior radiogram (shortly) after operation. An approximately normal conformation has been given the joint, although there is, naturally, a little "lipping" and blurring of the bone margins. The muscle sutures of phosphor-bronze wire show in this radiogram.

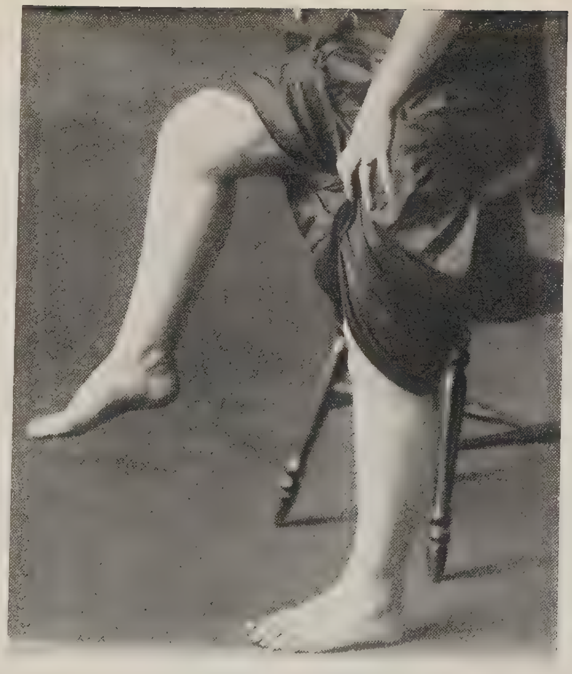


Fig. 304.—Arthroplasty for bony ankylosis of the right knee. The photograph, take nearly three years after the arthroplasty was performed, shows flexion possible to a right angle. Note the entire absence of any muscle atrophy in this vigorous, healthy limb.

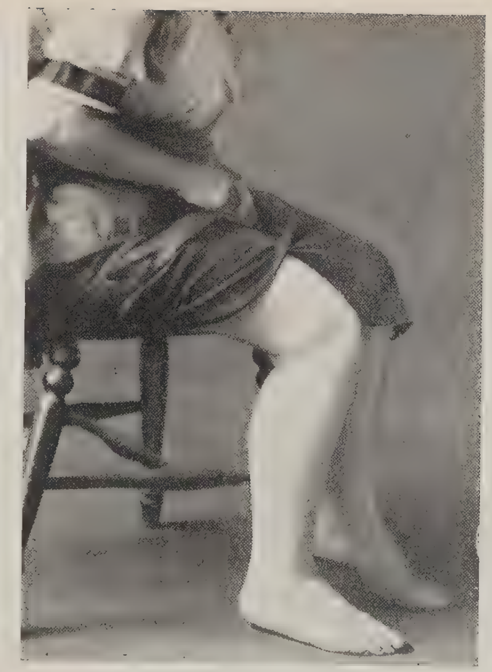


Fig. 305.—Arthroplasty for bony ankylosis of the right knee. The photograph, taken nearly three years after the arthroplasty was performed, shows flexion possible to a right angle. Note the entire absence of any muscle atrophy.

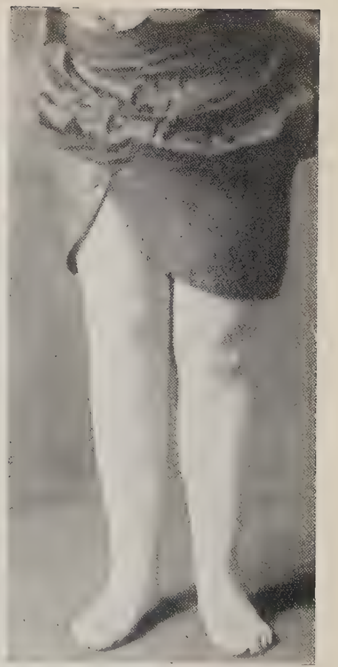


Fig. 306.—Arthroplasty for bony ankylosis of the right knee. The photograph, taken nearly three years after the arthroplasty was performed, shows the nearly perfect conformation of the right knee when in full extension. Except for the transverse scar, the appearance of the right knee seems almost perfectly normal. Note the entire absence of any muscle atrophy.

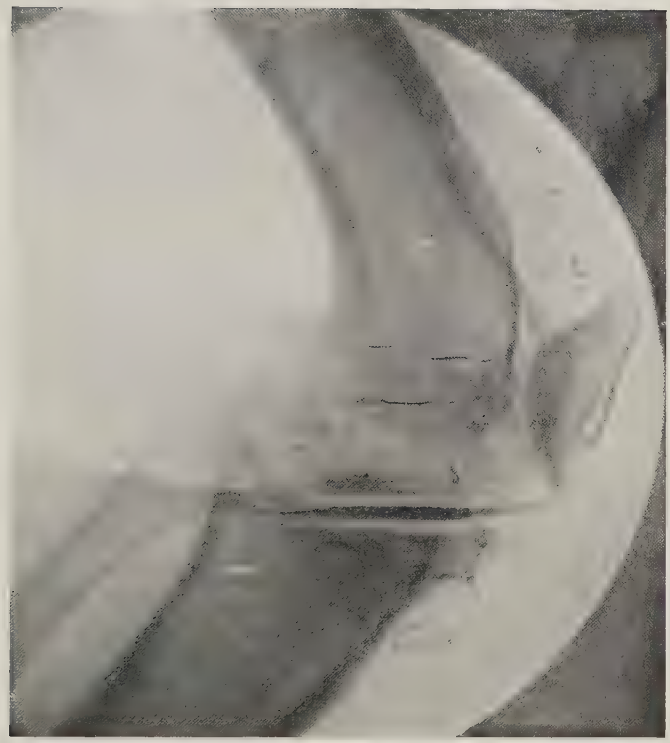


Fig. 307.—Arthroplasty for bony ankylosis of the right knee. Lateral radiogram made nearly three years after the operation. The patella is seen in profile, "turned turtle," and has, perhaps, taken on a more normal contour during the passing years. The muscle sutures of phosphor-bronze wire show in this radiogram and have produced no irritation or inconvenience in all the years they have been in place. Note entire absence of any "atrophy of disuse" in the bone.

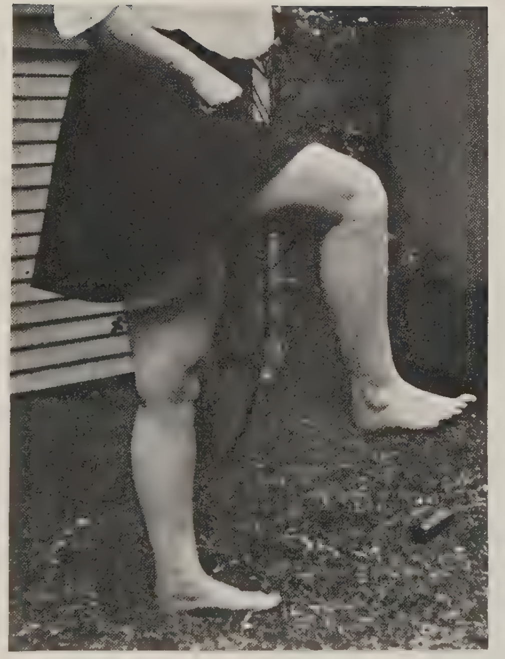


Fig. 308.—Recent Kodak picture of the same case.

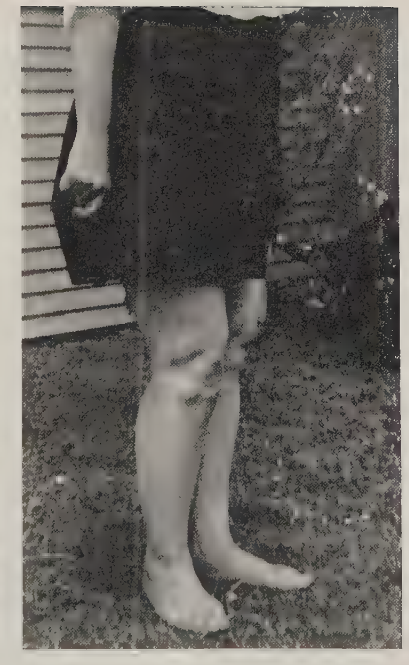


Fig. 309.—Recent Kodak picture of the same case.

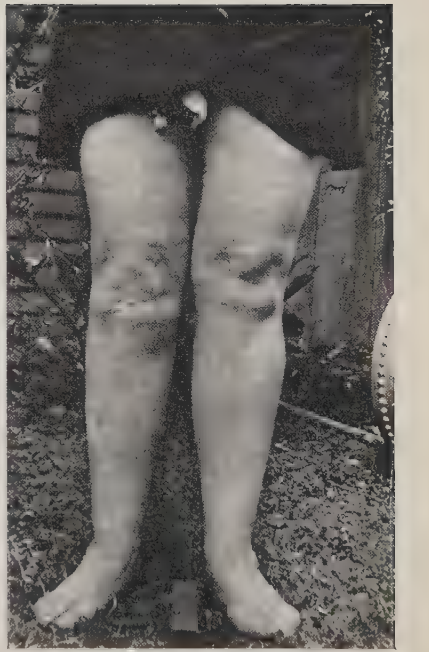


Fig. 310.—Recent Kodak picture of the same case. Note particularly the excellent and practically normal configuration of the new joint.

The Following Series of Drawings Illustrates the Steps in the Operation. Arthroplasty of the Knee for Bony Ankylosis, Several Cases of Which have been Reported Hitherto in the *Clinics*.

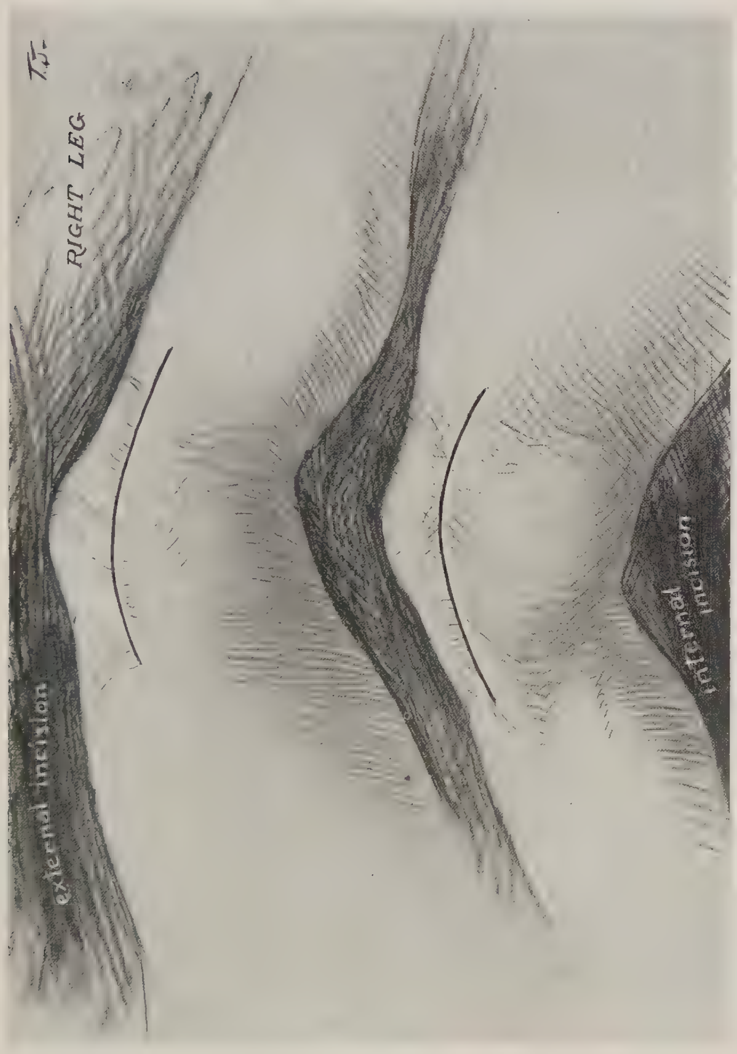


Fig. 311.—Internal and external curved incisions giving free access to the knee-joint on both sides.

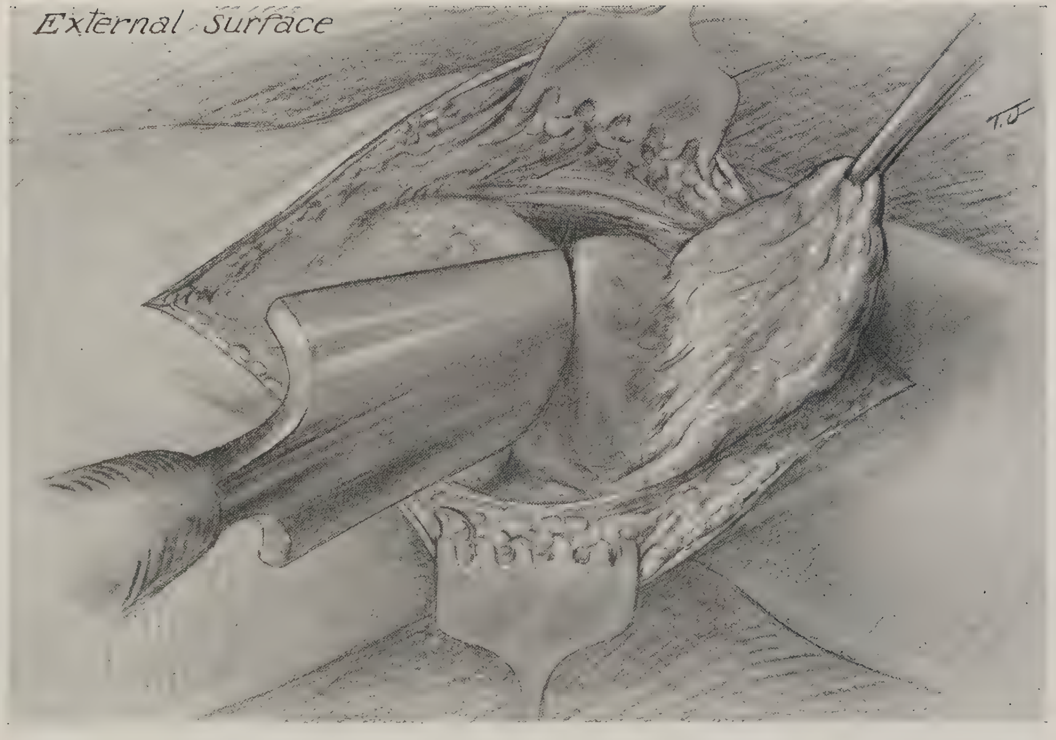


Fig. 312.—Freeing tibia from femur with curved chisel. Interposing flap prepared. Note that the curve of the chisel selected corresponds with the curve of the lower end of the femur. Although the pedicled fat-and-fascia flap was prepared in this case with its pedicle downward, it might as well have been prepared from the other direction.

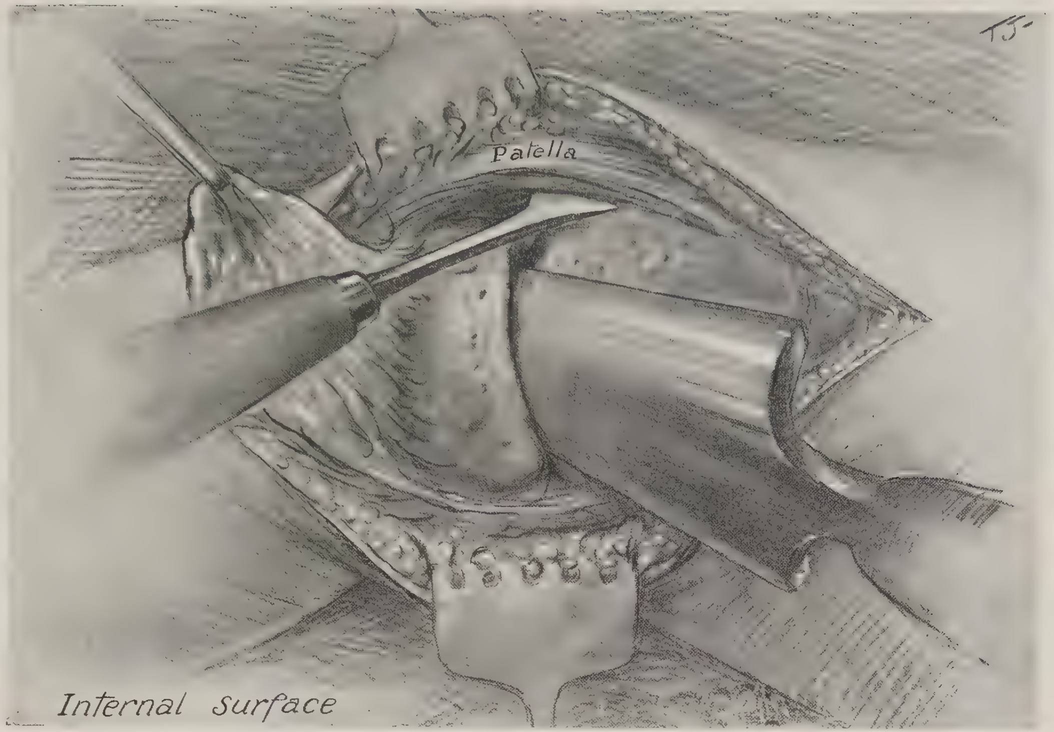


Fig. 313.—Freeing ankylosed patella from femur with "artist's" chisel. Interposing flap prepared. Freeing tibia from femur with curved chisel.

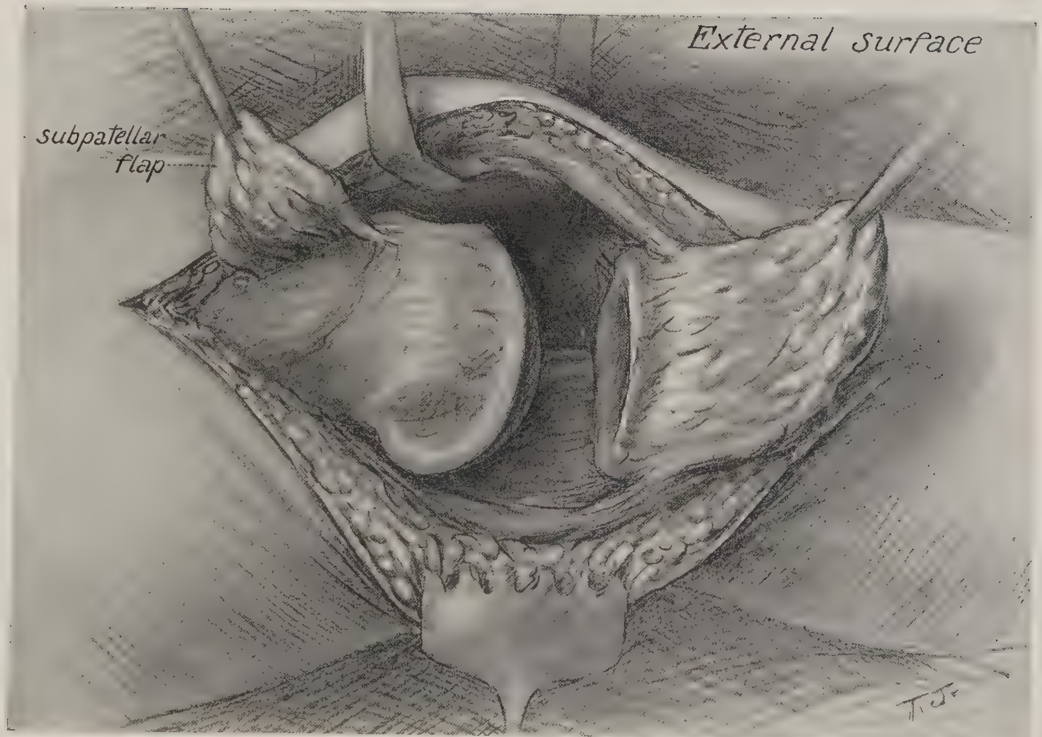


Fig. 314.—Articular surfaces of femur and tibia ready for insertion of interposing flap. Subpatellar flap prepared.

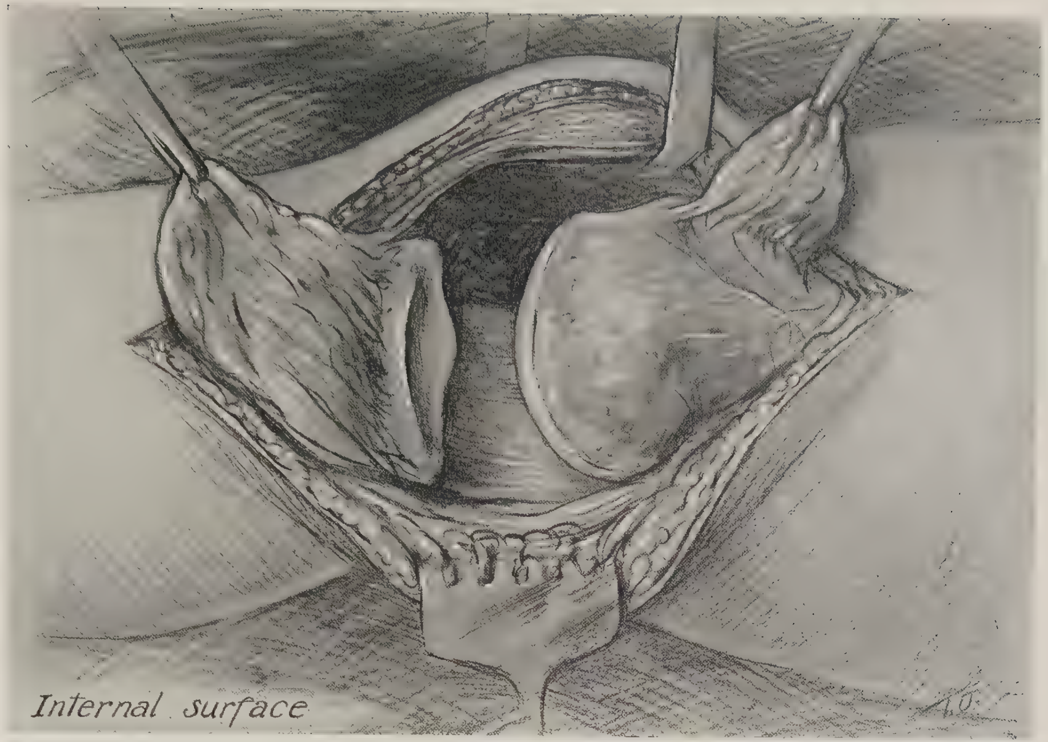


Fig. 315.—Articular surfaces of femur and tibia ready for insertion of interposing flaps. Internal subpatellar flap prepared. Note the width of the interarticular interval in the new joint—enough to accommodate the flaps easily, with something to spare.

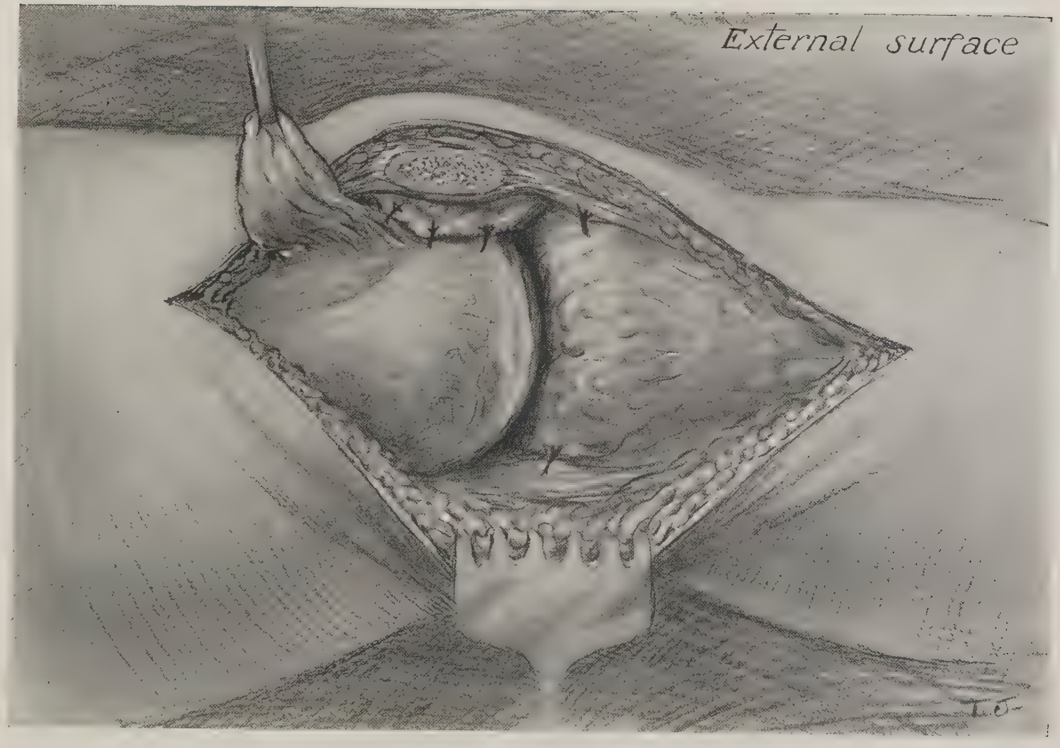


Fig. 316.—Shows external tibiofemoral flap inserted and outer edge of internal patellar flap sutured into place. External patellar flap prepared.

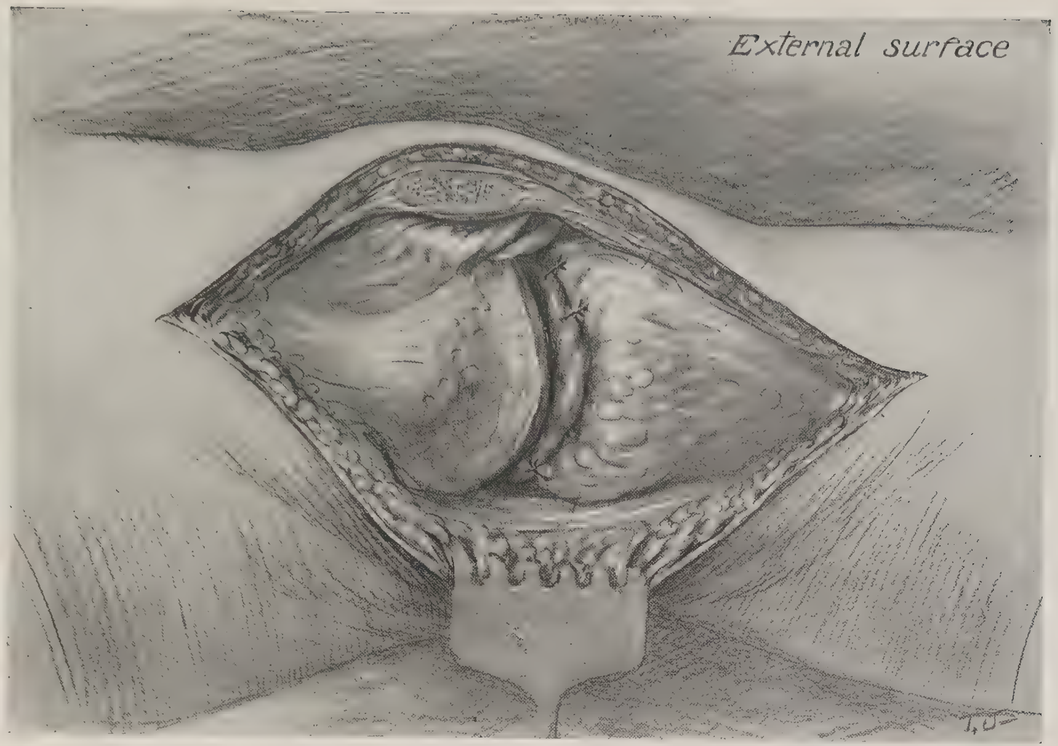


Fig. 317.—Shows the overlapping of internal and external interposing flaps.

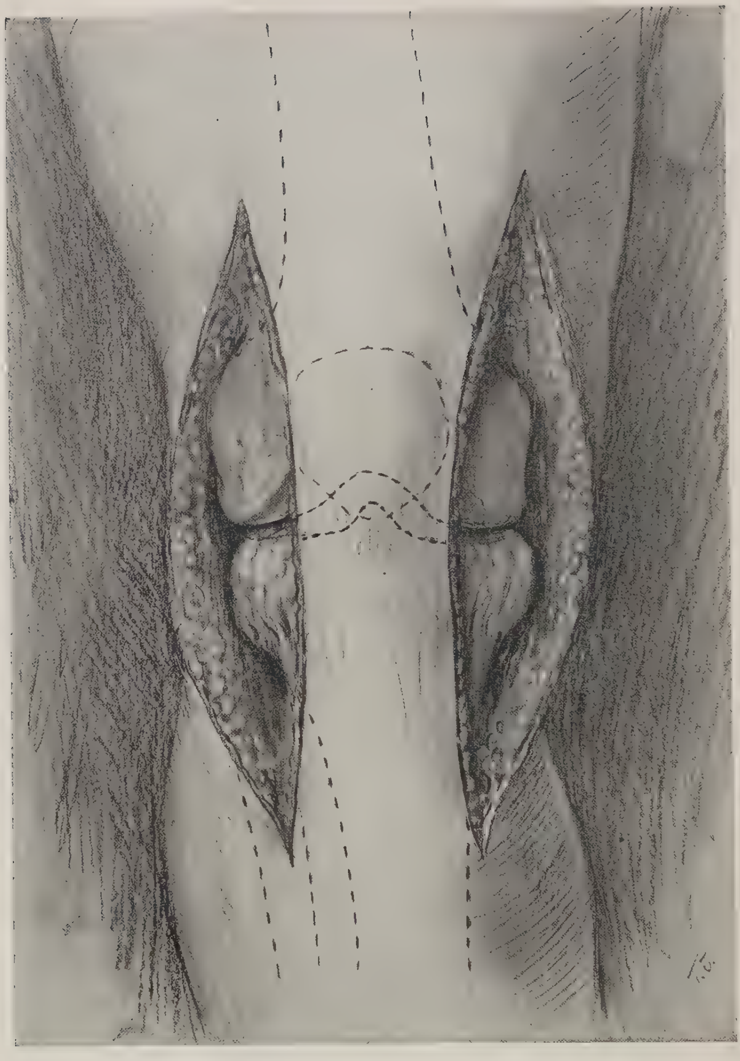


Fig. 318.—Schematic drawing showing the bone and anterior flap relations at completion of the operation.

THE FOLLOWING SERIES OF DRAWINGS ILLUSTRATES THE STEPS IN THE OPERATION, ARTHROPLASTY OF THE ELBOW FOR COMPLETE BONY ANKYLOSIS BETWEEN THE HUMERUS AND ULNA IN A POSITION OF COMPLETE EXTENSION, CASES OF WHICH HAVE BEEN HERETOFORE REPORTED IN THE *Clinics.*

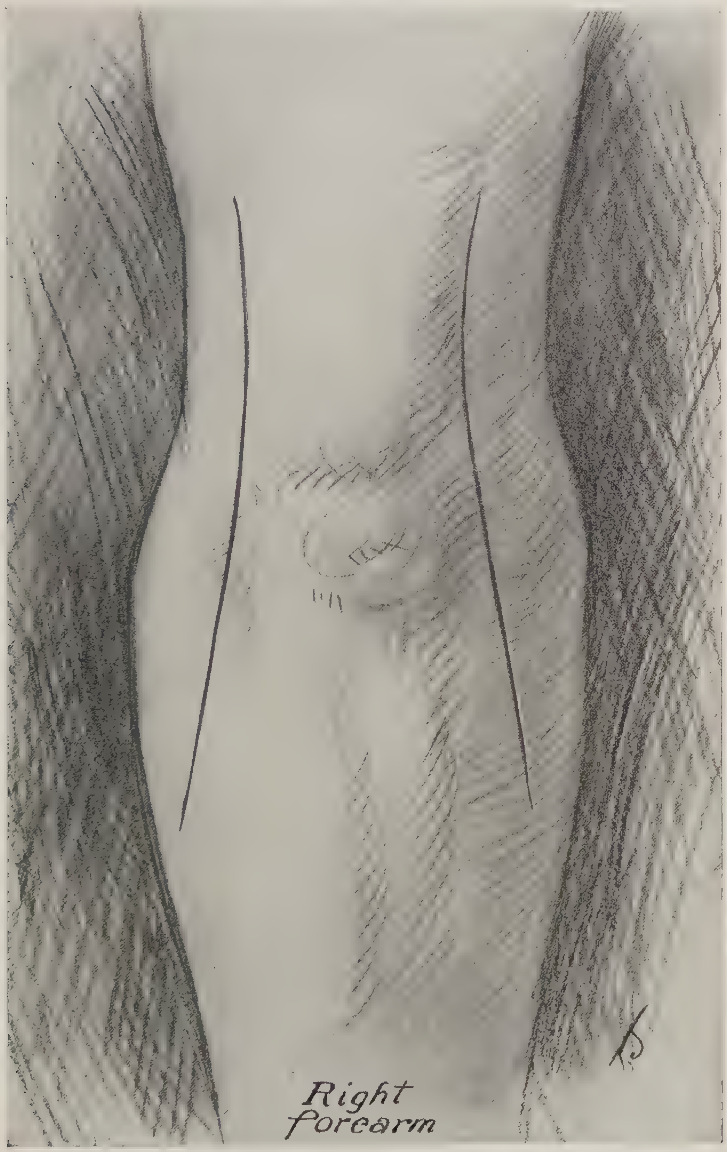


Fig. 319.—Posterior view of the right elbow, showing the location and direction of the two longitudinal incisions, one on the radial and one on the ulnar side of the olecranon.

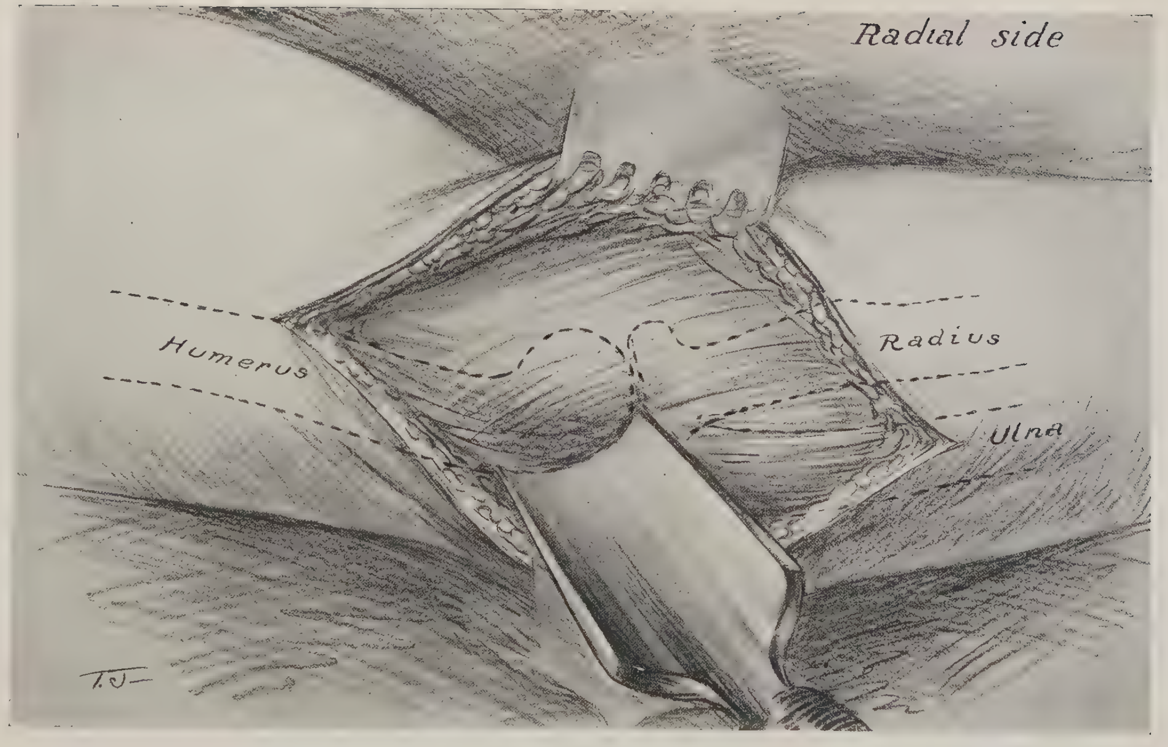


Fig. 320.—Exposure of the capsule of the joint from the radial side. The positions of the humerus, radius, and ulna are indicated by dotted lines. Note the direction in which the curved chisel is applied to separate the bones on the radial side of the joint. Note also that the curve of the chisel selected for the division corresponds exactly to the normal curve of the articular surface of the elbow-joint, thus reproducing in the artificial joint the exact contour of the original.

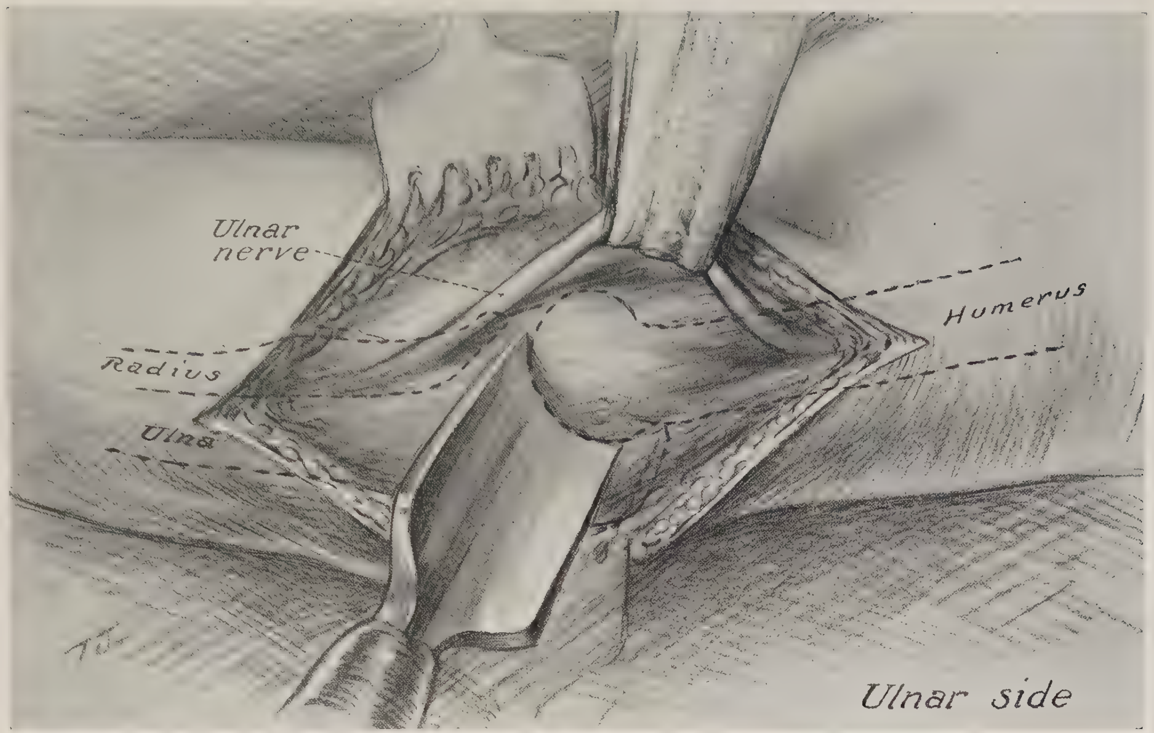


Fig. 321.—Exposure of the ulnar nerve and capsule of the joint from the ulnar side.

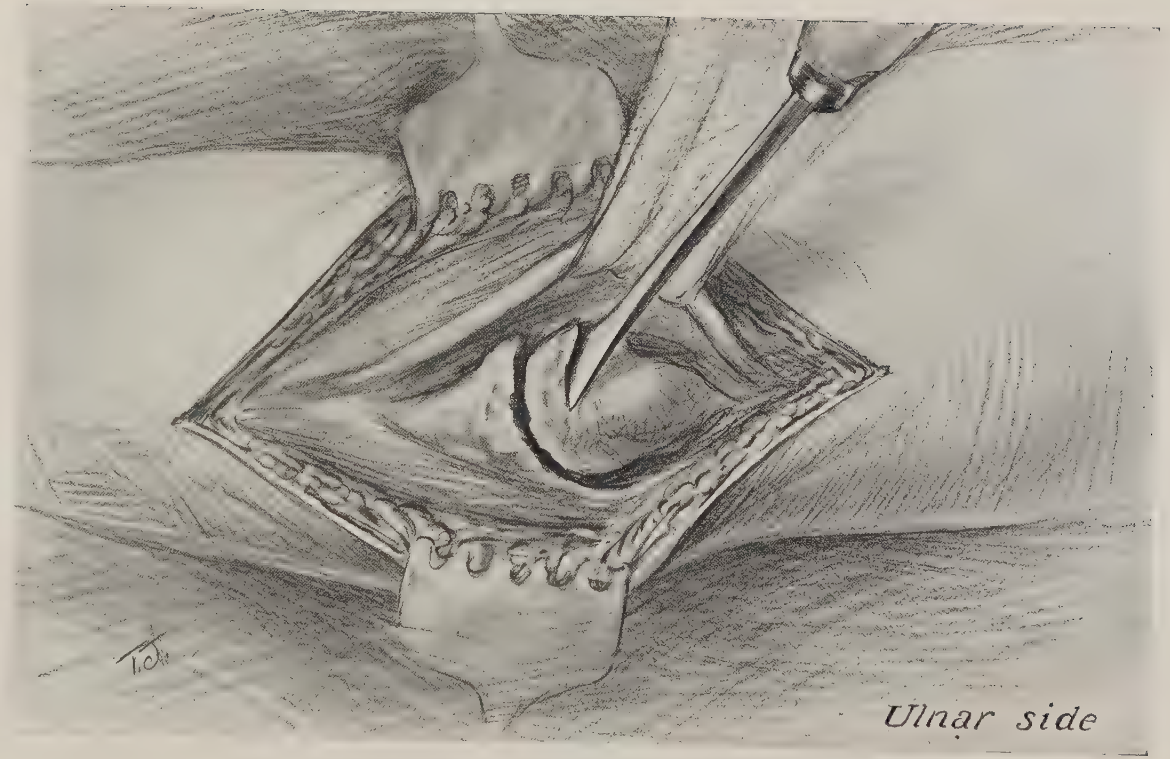


Fig. 322.—Removal of ulnar side of humerus.

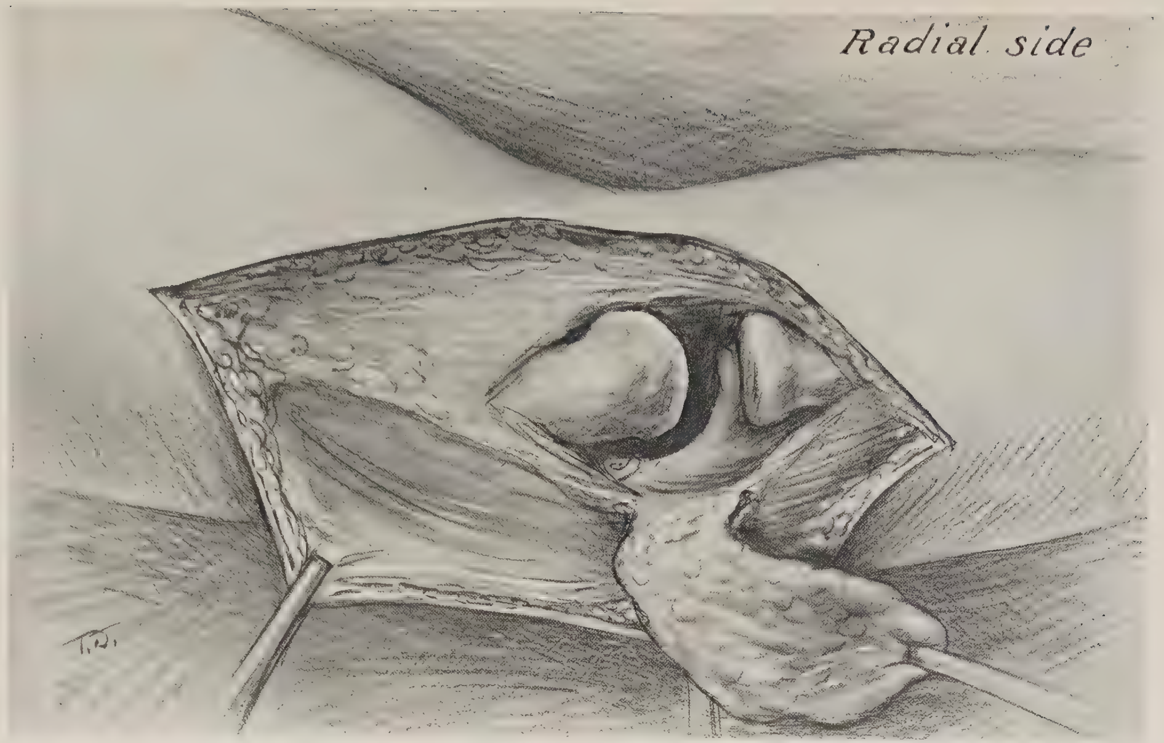


Fig. 323.—Pedicled fat-and-fascia flap prepared from the outer side of the arm for insertion into the joint from the radial side.

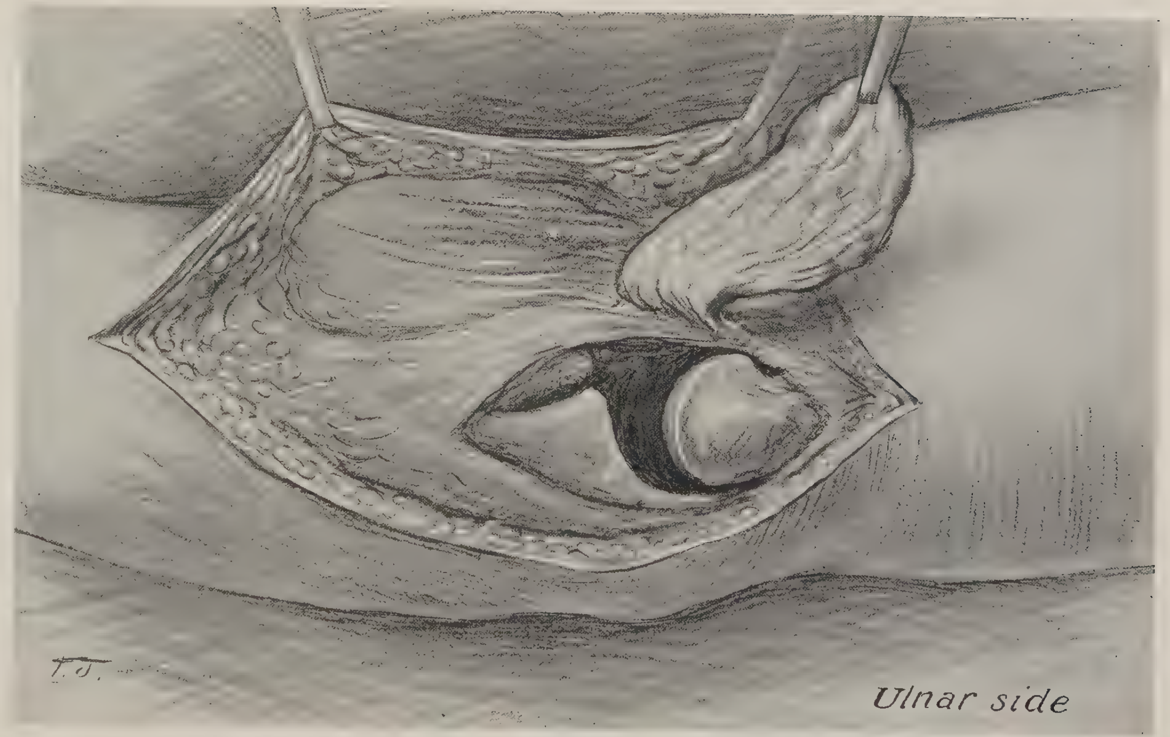


Fig. 324.—Pedicled fat-and-fascia flap prepared from the inner surface of the forearm for insertion into the joint from the ulnar side.

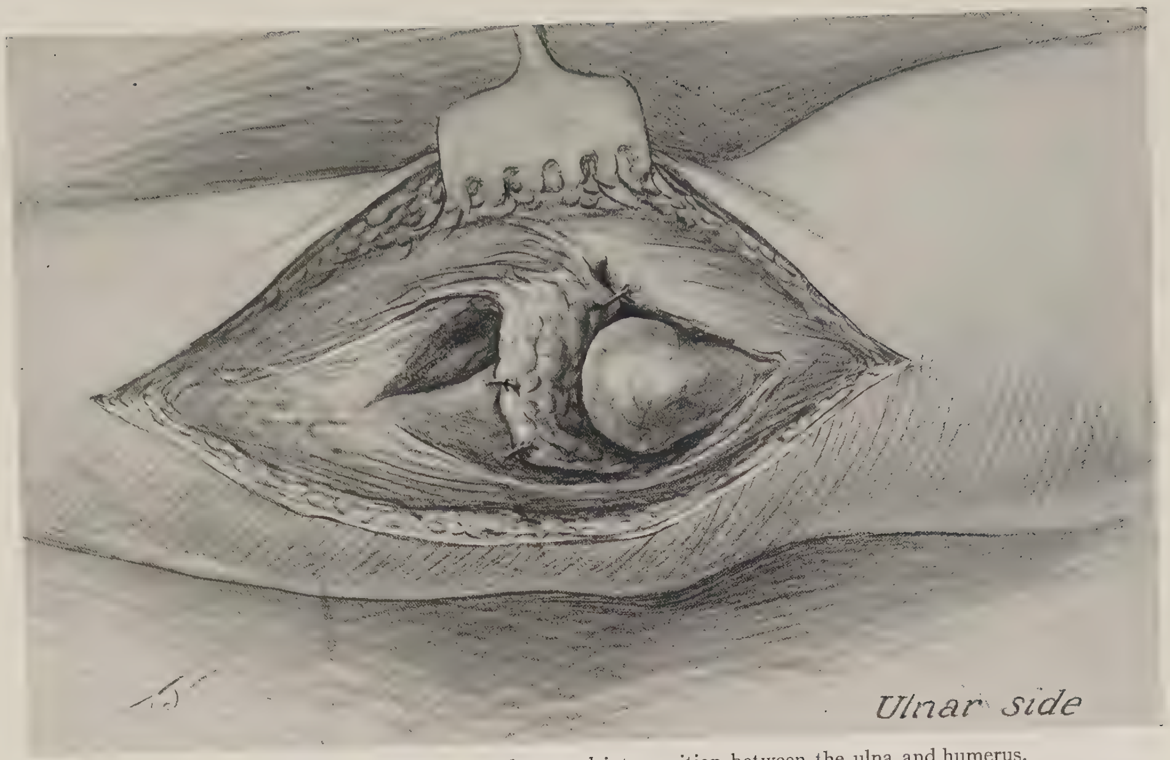


Fig. 325.—Ulnar flap interposed and sutured into position between the ulna and humerus.

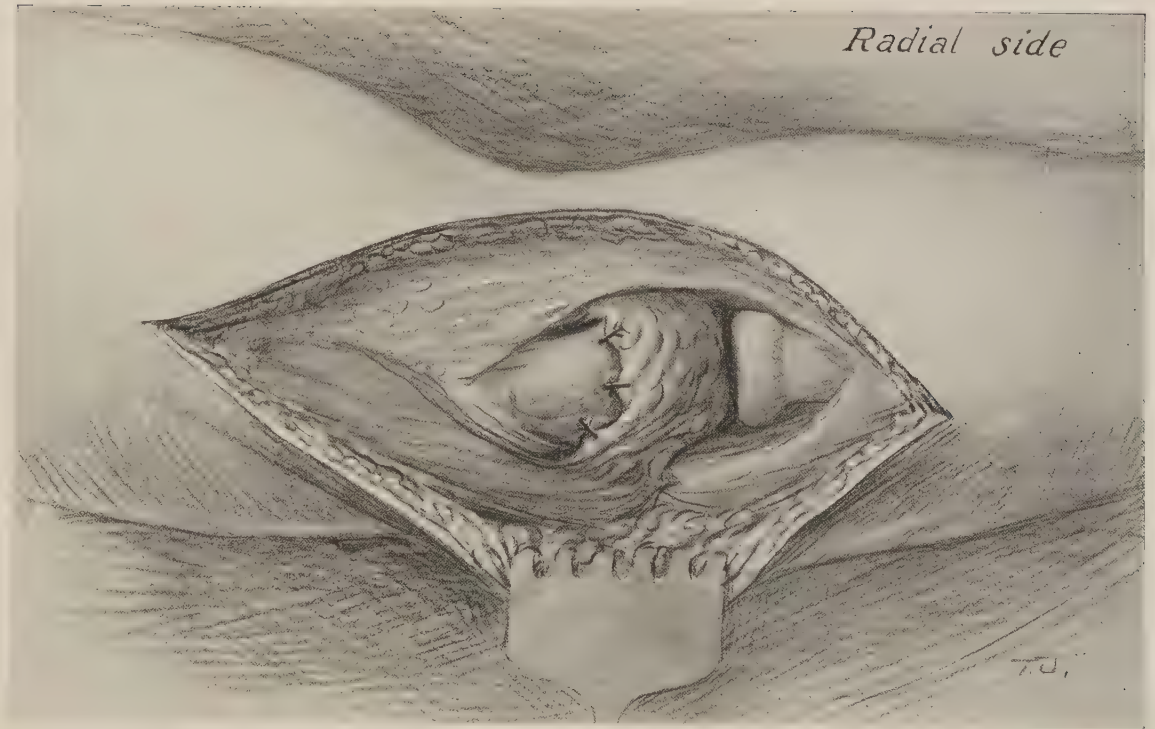


Fig. 326.—Radial flap interposed and sutured into position between the humerus and the radius and ulna. The deep stitches which fasten together the radial and ulnar flaps in the depths of the wound have not been shown in these illustrations.

HYPERTROPHY OF THE MIDDLE LOBE OF THE PROSTATE.—URINARY RETENTION.—PROSTATECTOMY

HISTORY

The patient, a married man aged sixty-three years, entered the hospital with the following history: For the past fifteen years he has had, at intervals, attacks of frequent urination accompanied by more or less difficulty in passing the urine. These attacks lasted usually about a week until some three years ago, when their duration increased to between three weeks and a month. These more prolonged attacks have been gradually occurring more frequently. Finally, six months ago,—in November, 1913,—the difficulty in passing urine and in starting the flow became continuous, and frequent urination became persistent. The patient had to get up three or four times during the night to urinate. For the past two months he has also had frequent burning pains in the bladder. He has never passed blood or pus in either the urine or the feces. He has never had any chills or fever. He has gained somewhat in weight in the last few months, he thinks. He was catheterized three times six weeks ago in order to evacuate the acutely retained urine.

COMMENTS AND OPERATION

Dr. Murphy (April 22, 1914): His first catheterization became necessary six weeks ago. The interval between micturitions at night has gradually become less and less over a number of years. His prostate even now is not greatly enlarged, although somewhat larger than normal. Recently he has come face to face for the first time with the real danger of his prostatic disease, namely, catheterization. He has had, judging by the history, transient infections of the urethra from time to time, but they did not produce sufficient inflammatory edema in the prostatic urethra to make catheterization necessary until six weeks ago. After three periods of acute retention necessitating catheterization six weeks ago, he has succeeded again in reëstablishing voluntary urination. The doctor who passed the catheter took every precaution at the time to avoid an infection of the urethra or bladder, and because he was catheterized only three times he has not acquired a urinary infection. If it had been 23 times or 53 times that the catheter was used, the chances are that he would now have a bladder infection. With repeated catheterization a traumatic urethritis is produced, on top of which a bacterial saprophytic infection is easily implanted. The patient is a physician, and he has kept a careful watch on the development of his affection. I think he has acted more wisely in the matter than most doctors do. He realized that when his enlarged prostate had arrived at the catheter stage, it was high time to adopt operative measures, and that it was highly dangerous to delay further until a suppurative cystitis with a suppurative urethritis, or a pyelonephritis was engrafted on the prostatic obstruction, constantly threatening him with death from sepsis and gravely complicating any subsequent operation on the prostate.

When I wrote my first article on the prostate, I insisted that the day on which the patient started to use the catheter was the day on which he should prepare for prostatectomy. I am happy to say that both the profession and the people are rapidly coming to that same conclusion. The results that have been obtained in such cases by the removal of the prostate fully justify that position. The average immediate mortality in prostatectomy is now down to about 2 per cent., including all the conditions for which the operation is performed. The mortality of prostatectomy undertaken after the establishment of catheter life is already very high and is on the increase. It is not likely that any series of prostatectomy cases will show a mortality much below 2 per cent., for the operations are performed on patients advanced in years, whose vital capacity is not at the maximum, and who are frequently sufferers from serious conditions, of which chronic nephritis, myocarditis, and arteriosclerosis are not the least. The patients with such affections have a minimal resistance against infections of all kinds, both local and general. Post-operative pneumonia is frequent, and ulceration and necrosis in the neighborhood of the bladder opening are sometimes severe and extensive, and may spread to the surrounding fascial spaces. The patients with advanced arteriosclerosis have particularly little resistance against infection.

Let us next consider the type of operation to be advised. When we started operating on enlarged prostates, we were advising the perineal route, and recommended the U-shaped incision. We had fairly good results with that method, but they were not at all as uniformly satisfactory by that perineal route as our present results are by the suprapubic route. The technic of the perineal procedure, no matter how slowly and carefully it is carried out, involves a greater manipulation of tissues over a more extensive and difficult anatomic field than does the suprapubic technic. In operating for any lesion on the neck of the bladder, one needs to bear in mind that in operations from above one opens the bladder through a relatively simple route—through its wall. By the perineal route, however, one opens the bladder through a highly complex anatomic zone, from the direction of its natural opening, it is true, but by far less simple a path. One might compare the suprapubic operation with an attempt to break into a house through one of its side walls, which is done by knocking out a few bricks with a pickax. The perineal operation, however, is like an attempt to enter the same house from the direction of the front door, but at an angle to it, so that steps, porch, and pillars must be cut away before the wall itself can be attacked. In entering by the perineal route, one is compelled to make just such an oblique approach to the urinary outlet, the functional capacity of which, therefore, is likely to be seriously interfered with. Since the prostate lies directly below the bladder mucosa, it is not necessary to disturb the parts surrounding it in removing it by the suprapubic route. All that is necessary is to divide the mucosa with the finger-nail and then peel the prostate out of its bed beneath the mucosa. Even with a careful and skilful perineal dissection, there are more dangers to be encountered by the lower route than by the upper. Freer, of London, does the suprapubic operation, and he, I believe, is more largely responsible for the present general adoption of the suprapubic route in removing the prostate than any other single surgeon. I remember visiting Freer once and examining the great collection of prostates which he keeps in glass cases. His earlier published articles did not give one a good idea as to the method he uses in taking out a prostate. One of the chief reasons why I visited him was to find this out. It was a simple thing which he taught me, but one of great importance. I have followed the method ever since. The point is this: Always start peeling out the prostate from the upper anterior edge, not the lower posterior. It makes a great difference in the ease with which the prostate peels out. We always use a sound in the urethra as a guide in our dissection. Pass the index-finger in along the anterior surface of the sound and tear through the vesical mucosa at the upper border of the prostate. One is able to peel out the prostate, as a rule, from the vesical mucosa, leaving the latter to flap back over the defect. The prostate, when removed, includes, of course, the prostatic urethra. This procedure is associated with much less danger of injury to the neck or *bas fond* of the bladder than is the dissection from the reverse direction. On opening the mucosa, one comes at once on the fibrous portion of the prostatic capsule. If one keeps carefully within that fibrous portion, the enucleation is made in the zone where the vessels are smallest and where they bleed the least. The moment one penetrates beyond that zone, one enters a vascular network which can be the source of a great hemorrhage at the time of operation, and, likewise, after operation.

I am passing the sound into the bladder as a guide. [Makes the usual curved transverse incision above the symphysis.] I am keeping as close to the symphysis as possible in carrying on this dissection, and, as I approach the bladder-wall, I am displacing the peritoneum upward and out of the field. Now I can feel the tip of the sound. Next we wipe the extravesical fat off the anterior wall of the bladder. The tip of the sound stands up in the center of the field, pushing the bladder-wall forward at its

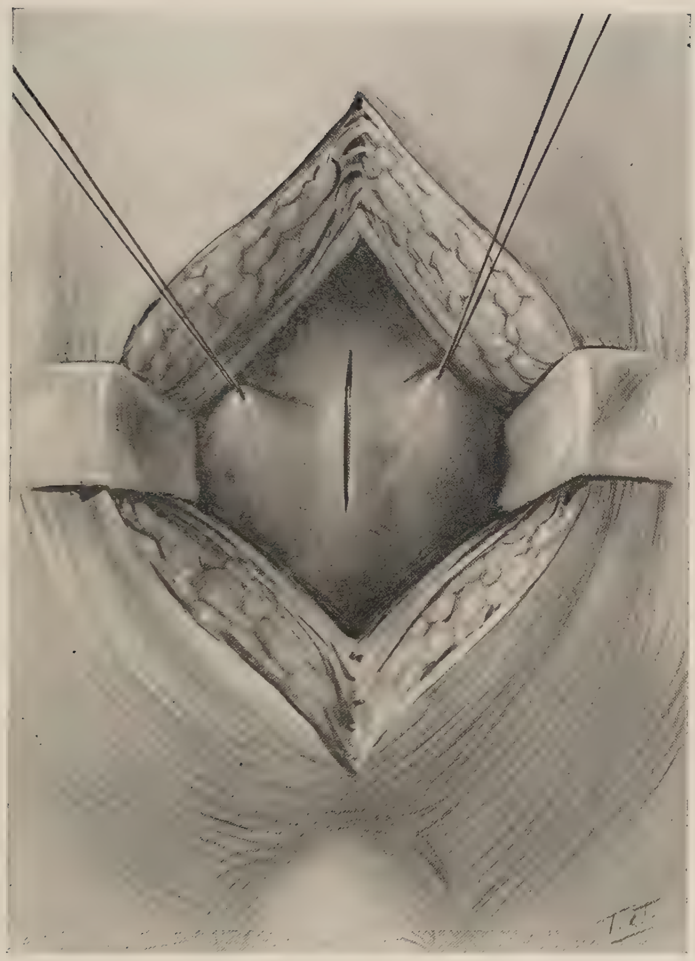


Fig. 327.—Suprapubic prostatectomy. Step I: The bladder-wall transfixed and held up by two linen "anchor" sutures. The incision made in the midline of the bladder between the two sutures and over the tip of a sound introduced through the urethra and pressed firmly against the anterior bladder-wall.

center. (See Fig. 327.) Before opening the bladder we pass the so-called "supporting" or "anchor" stitches through the entire musculature of the bladder-wall, one on either side of the prominence made by the tip of the sound. These stitches enable us to maintain control of the bladder all the time during the subsequent steps of the operation. The bladder-wall is divided between these two stitches over the tip of the sound.

[Dr. Murphy slips one end of a long rubber tube into the bladder opening, and lets the other end hang over the edge of the table.]

We siphon the urine out with this tube. When the urine is drained off we pass this self-retaining retractor into the opening. The retractor is to hold the bladder open, so that we can look into it and make a thorough inspection, as well as a digital examination.

There are no particular irregularities that I can feel along the bladder surface of the prostate (*i. e.*, no cancer nodules). The middle lobe is rather conspicuous, and appears to be the cause of the obstruction present.

[Dr. Murphy passes the fore and middle fingers of his left hand into the rectum, and with them presses the prostate forward, making it more accessible to the dissecting finger within the bladder.]

I am endeavoring to make a smooth enucleation of this rather firm prostate, using my right forefinger to separate the gland from its fibrous capsule.

[Dr. Murphy promptly completed the enucleation of the prostate. Pushing the dislodged gland to one side, he proceeded to make a bimanual examination of the operation site—the bed of the prostate—for remnants of gland, calculi, or other anomalies. It was found smooth and free from changes. The ensuing hemorrhage consisted of a mild degree of capillary oozing from the prostatic capsule. The loosened gland was lifted from the base of the bladder and out of the wound with a pair of gall-stone forceps.]

I always keep my finger in the rectum until the prostate has been grasped by the forceps and removed. This was not an easy case to handle. It was a rather difficult one, in fact, because of the firmness and relatively small size of the enlarged gland. The larger ones are always the easier to take out. We have learned that fact from experience. There is practically no bleeding at all to be seen in the bladder. Next we examine the bladder, both with the finger and with the eye, for other lesions, especially stones. Many times one finds calculi in the bladder in these old prostate cases, especially when there has been frequent or long-standing obstruction. Phosphate stones are the most frequent variety. There are no calculi or other pathologic changes to be

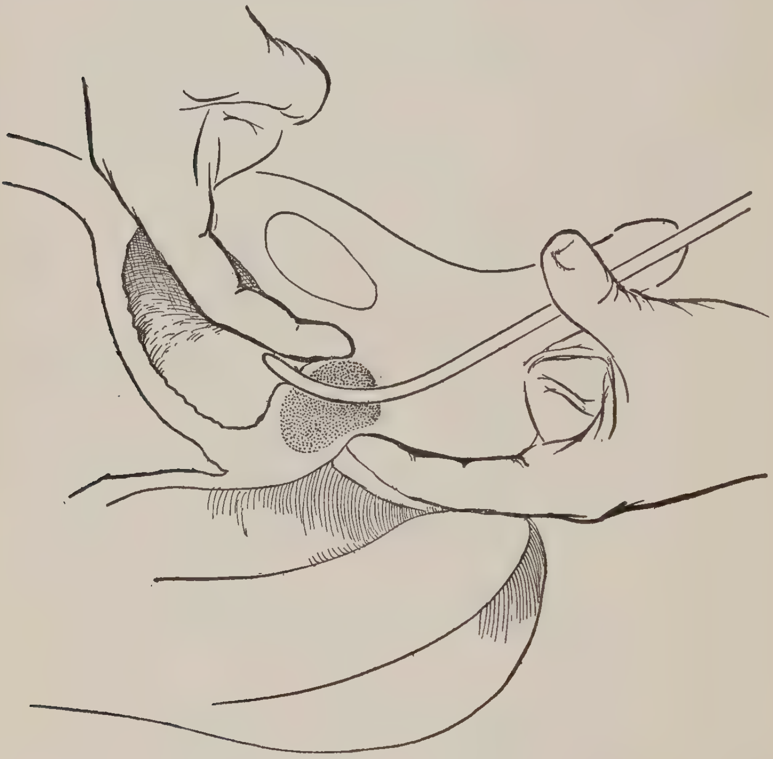


Fig. 328.—Suprapubic prostatectomy. Step II: Two fingers of left hand in rectum press prostate forward to meet index-finger of right hand, which begins the dissection at the upper margin of the gland. The sound in the urethra is drawn out until only its tip remains still within the bladder.

found, and no roughening or laceration of the edges of the prostatic wound.

In closing the bladder we use interrupted sutures, which lie entirely outside the bladder mucosa, being passed so as to include only the muscular layer of the bladder-wall.

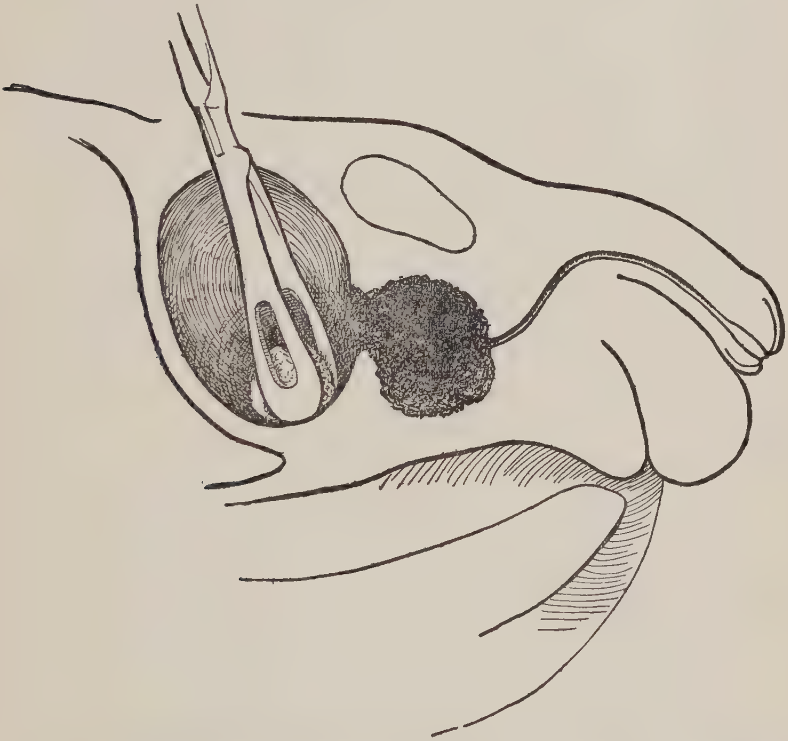


Fig. 329.—Suprapubic prostatectomy. Step III: The prostate, including the prostatic urethra, now dissected free, lies at the base of the bladder and is removed by forceps.

Let the record show that the prostatic enlargement was located practically entirely on the right side of the gland; that there was also a teat-like projection in the midline posteriorly, and that it was this projection which overhung the internal urethral orifice, that was the principal factor, probably, in his obstruction.

In resuturing the bladder-wall the anchor stitches which I inserted before opening the bladder are of great importance. In picking up the cut edges of the bladder we keep these original tacking sutures in view all the time. That is the advantage of having them there. We are never at a loss to know just where we are and what we are suturing. They are invaluable as landmarks, as one realizes best when attempting such an operation without their help.

[Completely closes the bladder incision with a double layer of Lembert stitches, being particularly careful not to pass any of the stitches through the vesical mucosa.]

Now that the bladder wound is closed, we withdraw these two anchor stitches.

[The muscle and fascia of the abdominal wall were then sutured with a running suture of catgut, and the skin with silkworm-gut figure-of-8 sutures, which also pick up the rectus fascia, and with horsehair.]

A permanent retention catheter will be immediately inserted into the bladder through the urethra. A small gutta-percha drain will be inserted into the abdominal wall down to the line of bladder sutures. The last catgut stitch through the bladder-wall was utilized to fasten the bladder-wall up to the sheath of the rectus, and thus keep the bladder from falling back and making a dead space which might fill with wound secretion, or with urine in case a leak should develop in our line of suture. That dead space is always a serious menace after bladder operations, and a phlegmon originating in it is a dangerous complication, because of the ease with which it spreads through the fascial spaces of the pelvis.

Let the record show that the prostate was completely enucleated; that the slight enlargement of the middle lobe was the important factor in the production of the urinary obstruction; that the greatest degree of enlargement was in the right lobe, but that the greatest obstruction was, as we suggested before operation, from the teat-shaped, overhanging middle lobe.

This gutta-percha drain goes down to the bladder, but not into it. We no longer use permanent suprapubic drainage of the bladder in cases like the present, with practically no hemorrhage from the operation site, and with no accompanying urinary infection. Formerly we used to drain all our cases from above, but we believe it merely retarded the progress of cases like the present one.

We attach this retention catheter to the penis, so that there will not be the slightest traction on it. It is fastened just over the foreskin. If the adhesive plaster fixation dressing is applied too tightly, a swelling of the foreskin may develop. We believe that in this class of cases we obtain better results by draining through a retention catheter in the urethra than through a suprapubic opening. Where we have more profuse hemorrhage we insert a suprapubic rubber tube in the bladder and drain through the operation wound as well as the urethra.

This was the kind of prostate that one can take out readily through the perineum. In my original paper on the subject I stated that a small prostate was particularly favorable for removal through the perineum. It is not the taking out of them which we fear. Like death, it is the after-effects, not the procedure itself, of which we are afraid, and the evil sequence of the perineal operation is loss of control of the sphincter. There is much better urinary control with the suprapubic operation.

In the first paper on prostatectomy written by Freer he said that he retained always the prostatic mucosa, both urethral and vesical. Because of this statement we went to the dissecting-room and tried to take out the prostate of cadavers and save the prostatic mucosa. It could not be done. We satisfied ourselves thoroughly on that point. In the very next article which Freer wrote he called attention to the extent of the urethra, about half an inch, that could be removed at prostatectomy without interfering with bladder control! From this statement we drew the natural deduction that he had come to the same conclusion as we.

This patient was fortunate in being a doctor and taking alarm in time. He did not wait until catheterization had produced serious urinary infection before submitting to operation. He will reap his reward by having an easy, short, and uneventful convalescence and a permanent cure. Will he also be potent?

Probably yes—at least, as much so as the average man of sixty-three years.

[Note.—Owing to a blood-clot lodging in the end of the catheter, the catheter had to be removed and a new one inserted at the end of twenty-four hours. No further trouble was experienced. The catheter was removed at the end of about a week and the patient left the hospital at the end of two weeks. The suprapubic wound healed by complete primary intention, the gutta-percha drain being removed at the end of twenty-four hours. After the permanent catheter was removed, the patient had apparently normal control of urination. He had no pain on urination, no difficulty in starting the stream, and no difficulty in retaining the normal amount of urine in the bladder. At the time of discharge his urine was perfectly clear, and the microscope showed no increase in the number of leukocytes. The patient ran no fever at any time. Catheterization showed no residual urine. Since leaving the hospital the patient has made no subsequent report to us, and we presume, therefore, that he is enjoying his usual good health.—Ed.]

IMPERFORATE ANUS

HISTORY

The patient, a female infant aged seven weeks, was brought to the hospital May 18, 1914, with the following history: The mother states that the baby was born at a normal labor and that it had bowel movements every day for the first two weeks of life. Since then, that is, during the last five weeks, it has been increasingly difficult to obtain bowel movements from the patient. On Friday, May 15, 1914, the baby had not had a bowel movement for nine days. Since then she has only had one slight movement. At the end of the first two weeks of difficult defecation, and after the bowels had refused to move at all for two or three days, the mother examined the baby and discovered that it had no anus. The doctor who was at once consulted examined the infant and stated that the feces came through the vagina. The baby appears to be otherwise normal in every way.

COMMENTS AND OPERATION

Dr. Murphy (May 18, 1914): That is all there is to the history—brief, but to the point. Now, then, doctor, where did the feces make their exit, and how often did the feces pass?

Intern: I do not know. The mother told me at first that the baby never had a bowel movement.

Dr. Murphy: How old is the baby?

Intern: Seven weeks.

Dr. Murphy: Then the baby *must* have had a bowel movement or it would be dead.

Intern: The mother states that it vomited everything it ate.

Dr. Murphy: That is not the point. The point is that the baby had bowel movements. It has had a bowel movement since it came into the hospital. It is well that you asked the mother again how many bowel movements the baby had. It is seven weeks old now and something must have come through its bowel or the baby would be dead by this time. We actually know from our own observation that since the baby came here she has had a bowel movement, small in amount, it is true, but unmistakably fecal. The intern's statement is correct that it vomited almost everything it swallowed.

On examination the vulva is found a little red and congested. The hymen is a little larger than usual, and the opening in it is wider than normal. There is an indentation, not deep enough to call a depression, about the position where the normal anus should be. The fecal material which appears comes out through the vulvar opening and through the vagina. Nothing comes out through the anal indentation. The question that one next asks is, Does the fecal stream come through the vagina, or does it come through the urethra? What type of congenital anomaly, in other words, has she?

These congenital abnormalities and defects in the development of the genitalia, rectum, and urinary apparatus are divided into certain distinct types. Particularly well defined are the rectal anomalies. They are:

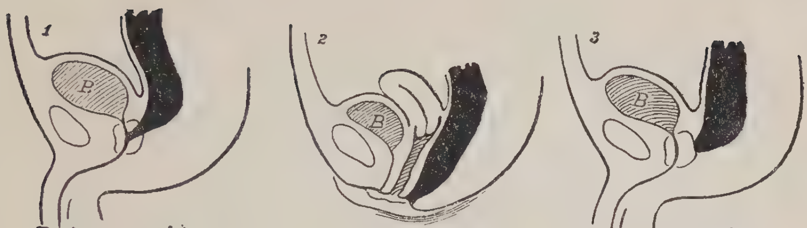
First: The undescended rectum, which remains high up in the pelvis or abdomen, and fails to come down far enough to become a part of the cloaca.

Second: The properly descended rectum, with total absence of an anal depression on the perineum, no effort being made to form a proctodeum by an indentation of the skin to meet the rectum.

Third: The rectum, which reaches down almost to the anus, with a slight anal depression present on the perineum, indicating that the rectum is just within or beneath the skin.

Fourth: The development of an anus and a considerable depression beyond it, with a cutaneous lining, but separated from the descending rectum by a shorter or longer interval, or connected with it by an impervious fibrous cord.

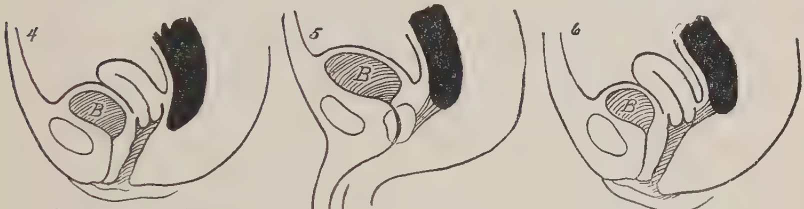
Fifth: The rectum in the male may open through the perineum, through the scrotum, or through the urethra or bladder, thus forming a congenital fecal fistula.



Rectum opens into prostatic urethra

Rectum opens into vulva

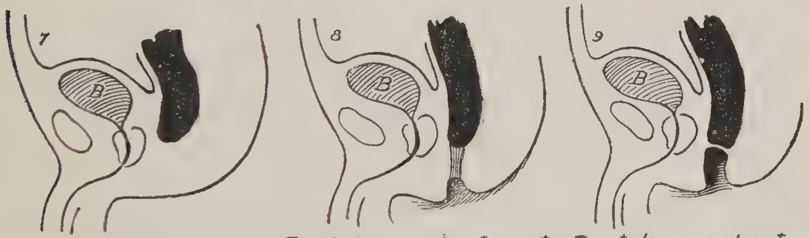
Rectum ending blindly at back of prostate



Rectum ending blindly at posterior fornix of vagina

Rectum attached to prostate by fibrous cord

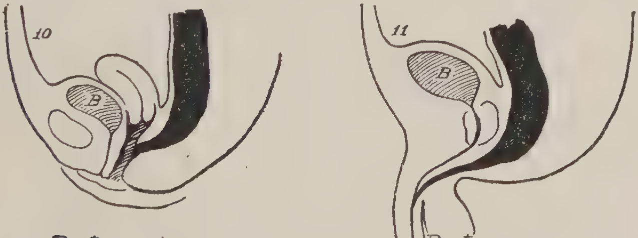
Rectum attached to post. vaginal vault by fibrous cord



Absence of proctodæum

Proctodæum and rectum not communicating but attached to each other by fibrous cord

Proctodæum and rectum separated by a thin membrane



Rectum opens into vagina

Rectum opens into penile urethra

Fig. 330.—The varieties of imperforate anus.

Sixth: In the female the rectum may open into the vagina, or, much less frequently, into the urethra or bladder. One may also find the urethra, bladder, and vagina *all* communicating with the rectum, so that urine as well as feces comes out through the rectum, so that fecal matter may pass out through the urethra, and urine mixed with feces pass out through the vagina, or through the anus, if the latter be patent.

We have not yet made an internal examination of the little patient, but we shall do so as soon as she is well anesthetized. We are considerably aided in analyzing the type of anomaly in this case by the statement of the mother that the baby's urine comes out perfectly clear. That means that there is no open communication between the rectum and the bladder. That is about all the information we have bearing on this subject, except that we know there must be a rectovaginal opening, as we have already stated.

[To examine the baby's vagina Dr. Murphy used a small-sized Kelly cystoscope, throwing in the light through a head reflector. The vagina was wiped clear of feces by the repeated use of cotton on an applicator.]

There is nothing to be seen in the vagina that looks like an opening into the bladder. Nothing comes out of the vagina that looks like urine; but fecal material and gas keep bubbling up somewhere along the posterior vaginal wall every little while. It is the presence of this fistulous opening that has been the life-saving feature in this particular case.

[Dr. Murphy then passed a flexible silver probe through the cystoscope and, finally, with its assistance, was able to locate the fistula rather high up on the posterior wall of the vagina. The probe, when introduced through this opening, ascended a considerable way without resistance and returned smeared with fecal material.]

Let the record show that the examination of the vagina through a Kelly cystoscope revealed no evidence of a vaginovesical opening, but did reveal the presence of the suspected rectovaginal fistula on the posterior vaginal wall. The vagina seems considerably congested.

[With the aid of the cystoscope Dr. Murphy then passed a ureteral catheter through the fistula up into the rectum, and then, beside it, he passed a flexible uterine sound so curved that its tip turned downward toward the terminal pouch of the rectum. It did not come down sufficiently far, however, to be felt from the outside, and, therefore, in the ensuing operation considerable dissection was necessary before the tip of the probe was located.]

There is a slight anal depression present here. There is also a little hole just inside this depression which looks as though it might possibly communicate with the rectum. [Dr. Murphy then tried to follow this opening with a fine silver probe, but was unable to find any communication with the rectum. In continuing his upward dissection, however, this opening was of some value as a guide, and it was in this direction that he proceeded and finally located the rectum.]

INTERN: The patient's doctor states that the bowels moved once each day for the first two weeks of life; then went nine days without any movement.

DR. MURPHY: Let the record show that we first passed a ureteral catheter and then a curved uterine probe alongside it, as a guide in our perineal dissection, through the vaginal opening into the bowel; that we then opened the perineum from behind, using the anal depression as the central point of our dissection, and gradually worked upward toward the tip of the uterine probe; that we exposed the posterior wall of the rectum; that we then exposed the lower end of the rectum, opened it, and divided its attachment to the wall of the vagina, closing the vaginal opening and the rectal opening separately; that the end of the rectum was approximated with catgut sutures to the skin, and the divided tissues behind the anus closed with silkworm-gut.

Let the record show that there were no feces present in the lower part of the rectum, that portion lying below the level of the rectovaginal fistula. The entire absence of feces in the lower rectal pouch was an odd feature of the case, because there was no obstruction to their entrance into this wide-open and directly adjacent pocket.

This is the first case of the kind that we have had this year.

It is a condition which occurs in one form or another in every 10,000 or 12,000 births. For that reason every obstetrician, pediatrician, and general practitioner should understand it and be prepared to meet it, because it is one of the things that can occur in almost any practice.

This baby had apparently good bowel movements for the first two weeks of its life nearly every day, and then they gradually became less and less in amount. Then, at last, it was noticed that the bowel movements were coming from the vagina, or, at least, through the vulvar opening. There was no anal opening and only a slight anal depression to be seen. The baby's movements gradually decreased in amount, so that, finally, for a period of nine days it passed no feces at all; but it vomited everything taken by mouth. What type of ileus was that? A mechanical obstruction without a strangulation, later developing into a practically complete obturation ileus, also without a strangulation and located at the lower end of the bowel. The absence of strangulation explains why that patient could go for nine days without a bowel movement and yet survive. If there had been a strangulation ileus present, the patient would have been dead in a couple of days, or in three days at the most. But because there *was* an opening to the exterior, although small, the baby was able now and then to get a little gas and fecal matter through to the outside, and that little makes the great difference between life and death in such cases. For nine days at a stretch she passed just a little gas through the vagina and no fecal matter at all; but at other times she has also passed a slight amount of feces through the vagina.

Congenital Malformations of the Rectum

I. Persistence of the original communication of the rectum with the cloaca.
  A. Males:
    (*a*) Ending of the rectum in the urethra at the lower end of the verumontanum, beyond the openings of the uterus masculinus and ejaculatory ducts.
    (*b*) Ending in the trigone of the bladder.
    (*c*) Ending at the external meatus of the urethra.

- (*d*) Ending at the apex of the prostate.
- (*e*) Opening into the frenum of the prepuce.
- (*f*) Opening into the under surface of the penis.
- (*g*) Opening into the raphé of the scrotum.
- (*h*) Opening into the raphé of the perineum.
- (*i*) Two openings:
  1. The proctodeum has grown in and opened into the rectum, forming an anus in the usual position.
  2. The cloacal orifice is prolonged forward and opens into the median raphé of the scrotum near the root of the penis.

B. Females:
- (*a*) The rectum opens into the navicular fossa of the vulvar cleft.
- (*b*) Opens into the vagina.
- (*c*) Vulva and clitoris are prolonged to form a urethra, and the rectum opens in its floor below the orifice.

II. Non-development or imperfect development of the post-allantoic gut.
- (*a*) Post-allantoic gut practically non-existent; rectum ends blindly at:
  1. Base of prostate.
  2. Upper level of vagina.
- (*b*) Post-allantoic gut may grow backward imperfectly and become separated from the prostate or vagina. It may end as a fibrous cord attached to either—
  - (1) the proctodeum,
  - (2) the site of normal anus.

III. Non-development or ill-development of the proctodeum.
- (*a*) The original membrane separating the proctodeum and the post-allantoic gut may partially or completely persist. The former condition results in a fibrous stricture, the latter in an absolute barrier.
- (*b*) The proctodeum may be feebly developed or there may be no anal depression at all. The nearer the rectum lies to the perineum, the better developed is the proctodeum; but a well-developed proctodeum does not signify a well-developed rectum.

IV. Congenital narrowing of the anus without complete occlusion.

The foregoing chart gives one a fairly good idea of the varieties of congenital conditions that can occur with an imperforate anus.

Unless one has in mind a pretty clear idea of the exact anomaly present in a given case, and, likewise, a clear mental picture of all the possible complications that may be met in handling that case, one is not justified in attempting to find the rectum, because, as one can readily see, great disaster can come to the patient through misguided surgical efforts. I cannot emphasize the great frequency of such disasters better than by citing the Cripp table, showing the types of operation performed for this condition and their results. He collected 100 cases, with a 50 per cent. mortality. A 50 per cent. mortality is too great a mortality for this condition in the hands of an operator who has a clean-cut mental picture of what he expects to do and what it is possible for him to find during his exploration in the individual case.

In making the new anus we fixed the rectum to the margin of the skin at the place where the sphincter muscle seemed to be located. We believe we have not interfered so materially with the sphincter as to affect its functional capacity seriously. If we have, the patient will subsequently suffer from incontinence; but we hope we have been able to avoid injuring it. We went, in our dissection, between the two layers of muscle which make up the levator ani and, we believe, we passed between the two halves of the sphincter ani. This patient has run no risk to her life in this operation. There is a little possibility of an infection from above, but not from below. We knew before we started that we were going to find the rectum in a fairly definite location, close to the posterior wall of the vagina; and with the uterine sound we maintained a definite guide to our procedure during the entire operation. In operating these cases one must have a fairly clean-cut picture of the situation in one's mind before making the incision; because, in some cases, one incision may be in the abdomen and the other incision on the perineum or elsewhere, and one cannot elongate even in case of dire necessity one of the incisions to meet the other. Therefore, the incisions must be planned on an accurate knowledge of the conditions present, and one cannot make an accurate diagnosis in this line of cases without a clear understanding and a definite mental picture of all the pathologic conditions which may occur.

SECOND OPERATION

Dr. Murphy (July 3, 1914): We let this patient go home shortly after the operation, when the rectal wound had healed, as it did promptly, and without any complications whatever. We thought we had made a good-sized anal opening, and we had; but after the patient returned home it began to contract. The operation scar was annular, and it tightened up rather rapidly. We must dilate it again and put in a rubber tube until it stays dilated. We were successful in saving the sphincter, just as we hoped. In saving the sphincter, however, we made this annular scar about the anus. The anal opening at present appears completely closed, but the rectovaginal fistula is still leaking. If we get this anal orifice fully open, we believe that the fistula will close completely. If the fistula does not close of itself, we can still close it by an operation. That rubber tube which we have inserted in the anus will be kept in continuously, and the anus will soon accommodate itself to it.

Let the record show that the anal opening, because of the annular scar about it, had closed down to pin-point size, and was so narrow that it would not admit the small probe; that after dilating it a rubber tube was inserted and held in place by the dressing; that we put a safety-pin on the tube to keep it from slipping up into the rectum. We did not use a silkworm-gut stitch to hold in the rubber tube, as we often do in drainage cases, because the contraction of the sphincter may pull on the stitch and make the stitch cut through muscle and skin.

[Note.—The patient went home the same day and has not reported since.]

THE USE OF RADIUM AND THE *x*-RAYS IN THE TREATMENT OF CANCER

A Talk in the Course of Operating on a Case of Carcinoma of the Breast

VISITING DOCTOR: Do you think the *x*-ray is of any value in the prevention of a recurrence of a breast carcinoma?

DR. MURPHY (September 2, 1914): Yes, I do. Do I *know* it has any advantage? No. Do I *think* so? Yes. We advise it, we order it, and we believe it does some good. Its retarding action on the development of a carcinoma is due, we believe, to the fact that it is capable of stimulating cicatrization or connective-tissue formation around the malignant cells to the degree of strangulating them. On these malignant cells which have escaped from the primary focus into the neighboring tissues, we believe that the *x*-ray has some effect. We use it on practically all our carcinoma cases after operation, and we use a relatively hard tube in giving the treatments.

We have not had much experience yet with the Coolidge tube, but every present indication points to the conclusion that Coolidge's invention is one of the greatest recent advantages in *x*-ray engineering and is going to be invaluable to us in the treatment of deep-lying and inoperable malignant tumors. We never use the *x*-ray on a case as a substitute for an operation. We use the *x*-ray occasionally on inoperable cases. We use it a great deal, however, in the hope of preventing recurrence after we have removed by operation all the visible tumor. While I was abroad this summer I took the opportunity to go into all the details of the present status of radium therapy in both the Radium Institute and the Cancer Hospital in London. The surgeons in charge of both of these efficient modern institutions, scientists of great authority who have attacked the problems of cancer treatment from every angle, never speak of radium as a cure for deep-seated cancer; that is, they never advise it in any case that is still operable. So strongly are they intrenched in this opinion —we seize naturally on military terms in these days—that *not only do they not advise radium in these cases, but they will not administer it to such patients as are still in the operable stage of the disease.* In other words, they will not be parties to the crime of suicide—which a cancer patient commits when he omits knowingly the necessary operation during the curable stage of his malady. In the operable varieties of cancer these Londoners believe that radium has a strongly inhibitory effect on the growth of the carcinoma, and that it materially retards the speed with which the tumor destroys the patient; that the patient lives longer and lives more comfortably with the radium treatment if his is an inoperable case.

When I was in Leeds, visiting Mr. Moynihan, I saw a large inoperable carcinoma of the side of the face just anterior to the ear—exactly the type of a case that came to my office yesterday, and that I may be able to show you here in clinic within a few days. For this patient Mr. Moynihan used not the pure radium, but the radium-sensitized material which comes in a little tube sealed in glass. He punctures the cancer tissue and plants this tube in it. Mr. Moynihan believes that this method is more effective in the management of large tumors than the use of the pure radium itself. Captain French, who has charge of the Radium Institute in London and is a master of the subject of radium therapy, holds the same view as Mr. Moynihan. Mr. Moynihan told us that this cancer of the face stood out from one-half to three-fourths of an inch above the level of the skin when the patient began treatment. When we saw him the tumor had practically disappeared, and one could tell there had been a lesion there only by close examination. The patient had had three implantations of radium-sensitized tubes in the growth. I spoke about this case to Captain French, and he said, "Yes, that is just what radium will do in the first treatment; but that growth will recur, and the next time that the radium is used it will have little or no effect on the tumor."

That is a sad outlook, but it appears to be the truth of the matter. While radium in the primary application may destroy the cancer to a great degree and retards its further growth, it does not seem to have the same effect on the tumor in subsequent applications. In reply to my question as to the cause of the destructive effect of radium on these malignant growths, Captain French stated that its action is that of excessive stimulation of the tissues exposed to its action; that it stimulates the cells to the degree of necrosis; that it actually cauterizes the surrounding neoplasm. Following the death of the tumor tissue comes, in its place, an excessive production of connective-tissue cells, which tend to strangulate such malignant cells as may have escaped the action of the radium. That is the theory of the destructive action of radium, as Captain French tells it.

In cases of inoperable carcinoma of the rectum, radium has a striking effect. It stops the bleeding, it stops the pain, and it stops the tenesmus; it retards the course and spread of the disease; but it does not cure. The place where it has the most striking effect as a palliative measure is in inoperable carcinoma of the uterus. In these cases they implant the sensitized tube into the mass of tumor tissue at the cervix. The subsequent result looks more nearly like that in a case treated by zinc chlorid packing, which was brought out by A. Reeves Jackson of this city thirty years ago, than anything else with which I am familiar. It produces a local necrosis of the tissues, somewhat elective toward the cancer, but not entirely elective, because, in cases of which we were told in London, there were a number of cases of fistula of the bladder, and a similar number of cases of fistula of the rectum following its application to the uterus. The significance of this fact, I take it, is that the radium necrosis extends beyond the limits of the cancer. The subsequent course of these cases is that the further development of the growth is markedly inhibited and that the type of tumor changes from the rapidly fatal medullary form to the slower growing scirrhus. Recurrence is inevitable, but death comes relatively slowly in the radium-treated cases, and the discomfort caused by the tumor is decreased by lessening the discharge from it and diminishing the dangers of saprophytic infection and hemorrhage.

That is about what one may expect from the use of radium in columnar-celled adenocarcinoma. In the squamous cancers, such as occur in the mouth, they do not advise radium at the Institute. They have had just about the same degree of success with these cancers as the surgeons. Any one who has had experience in operating on mucous membrane carcinomata is very happy when these cases go to the other fellow, because he knows that the cancer is going to come back, in all probability, and terminate fatally, no matter what is done. This opinion of the action of radium on mucous membrane cancers is also supported by Abbe, of New York, who has had quite an experience along this line. Just before I went to Europe this summer a man came to me with a carcinoma of the mouth, beginning at the angle of the jaw. I sent him to Abbé for radium treatment, but Abbé would not treat him; he said it was no use, and sent him back to me. Captain French, on exactly the same grounds, refuses to treat these cases in the Radium Institute of London, because he knows from his experience that they are certain to terminate fatally.

In the superficial varieties of carcinoma ot the skin, the type of carcinoma that has been cured by the application of the *x*-ray, I think that radium has a more rapid action, is more easily controlled in its application to small lesions, and its activity can be more definitely limited than the *x*-rays, and appears to be more effective because it is superficial in its action. In the treatment of the angiomatous type of birthmarks, radium is a splendidly effective therapeutic agent. In the treatment of keloids it is also a fine thing.

That seems to be the present therapeutic position of radium. Exactly the same opinion was voiced by the physician in charge of the radium section of the London Cancer Institute, a man of great experience and high scientific attainments. He showed us every possible courtesy, and it is with great regret that I must admit his name has escaped me for the moment. He agreed entirely with the conclusions of the workers in the Radium Institute.

We have, therefore, from the use of radium, gained no important additional data for the cure of cancer. We must put the radical treatment of cancer back on the same surgical basis where it has so long rested. Cancer is a surgical lesion from its very beginning. It is at first purely local in its manifestations and should be removed at the earliest moment, while it is still a local disease. The colossal force of numbers shows in cancer statistics that, no matter what our theory as to the nature of the malady, a larger percentage of cancer cases remains well when the tumors are taken out early than when removed late in the disease. That is the present clinical proposition, and it is the clinical standpoint in these cases which is all-important.

That radium is of some advantage to hospitals in the management of inoperable cases of malignant tumors we think is true. It is of some advantage in a large city in treating the inoperable cases, because these patients prefer to live at home, and, while under radium treatment, are able to do so. An operation takes them to the hospital. The *x*-ray, properly applied with a hard tube, gives better results than *x*-ray treatment formerly did, because we are thus able to prevent practically entirely the burning of the skin, and we are able to take out practically all the alpha and beta rays. Also, when we are able to eliminate the alpha and beta and leave only the gamma rays, the latter seem to have greater effect in stimulating the production of connective tissue around the irritated cancer-cell nests. We hope that when the Coolidge tubes are made in greater quantities, and their price lowered to meet the means of the many hospitals and physicians who now use the *x*-rays in treatment, some further advance in the treatment of deep-seated, inoperable cancers may be made. The Coolidge tube is so constructed that it automatically eliminates the dangerous and useless alpha and beta rays, and produces only the gamma rays. Moreover, it produces them in great quantity, for the tube can be run day in and day out at very high amperages without deteriorating or discoloring the tube, and, what is still more important, without altering the tension of the tube and the character of the rays produced.

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